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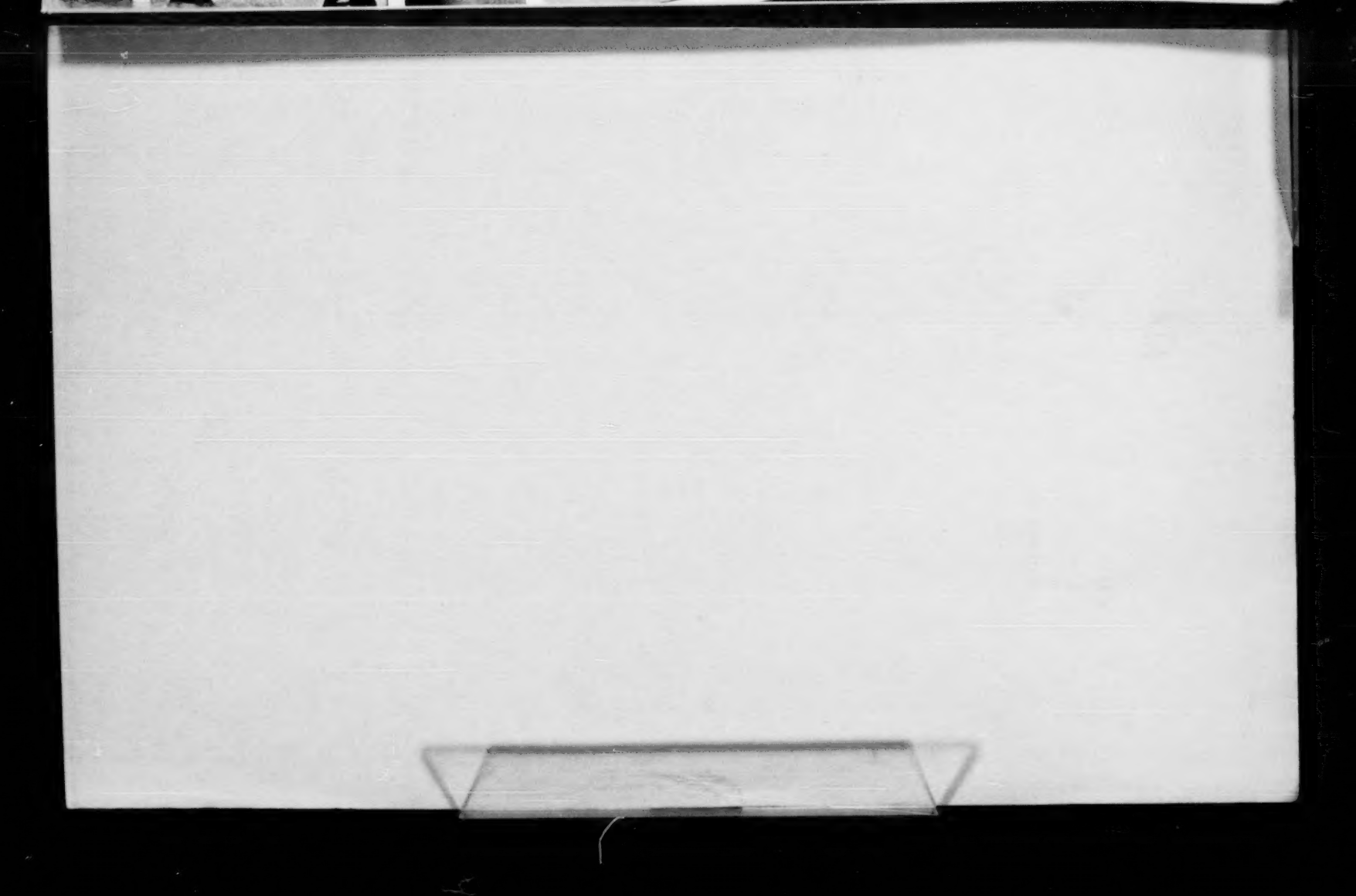

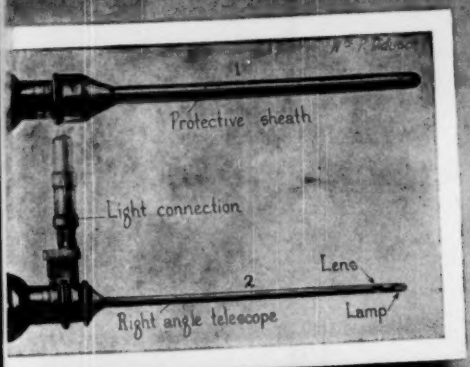

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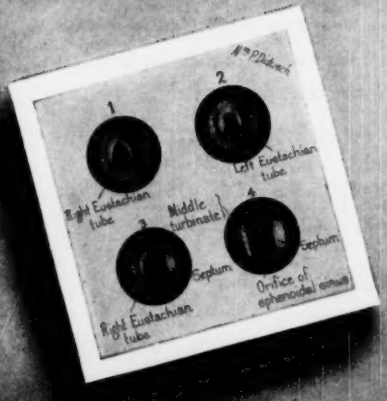

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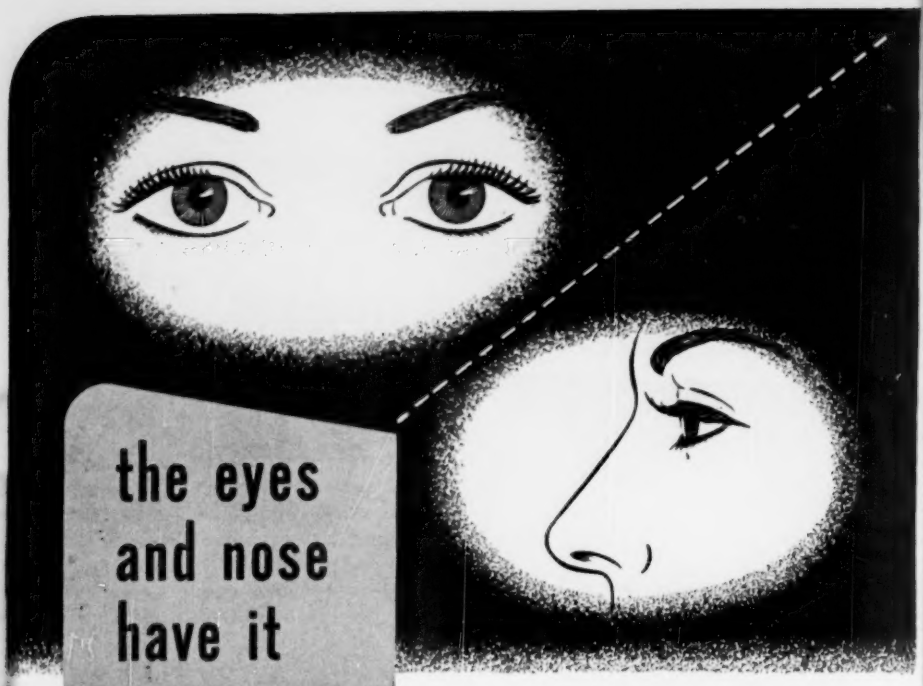

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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HEARING DEFECTS ARE OFTEN MISSED

Generally overlooked is the onset of hearing difficulties. Even the doctor, his attention directed toward a diagnostic problem seemingly unrelated to hearing, may not be aware that his sensitive patient is hiding a hearing defect; that his cantankerous patient is merely venting an irritation generated by not quite hearing what people about him are saying; that another patient is tense because of constant effort to hear, without being aware of his trouble; that a sullen patient may not answer because he cannot clearly hear the question put to him. To the partly deaf, the world is a place where every spoken word seems just beyond reach.

Too often people are prone to consider a person either deaf or not. The wide twilight zone of those who are just deaf enough to be vexingly handicapped is too often neglected.

Perhaps this neglect is due to the terms employed in speaking of these people. Regardless of the degree of hearing loss, whether slight or total, they are popularly grouped together as "deaf."[1]

"Deaf" and "deafness," the American Otological Society declared, are terms to be applied only to those whose hearing loss is so severe as to make it impossible for them to carry on their daily life. "Hard-of-hearing" is the scientific term to use for those with lesser hearing defects.[2] These definitions, however, are academic rather than practical.

THE PUBLIC MISUNDERSTANDS THE HARD-OF-HEARING

The misconceptions about those with impaired hearing are often ridiculous and generally cruel.

Such people are "more or less ostracised,"[3] and yet—so inconsistent is human thinking—it is considered that their affliction is "relatively trival"; that lip reading is a satisfactory substitute for hearing[4]; that they have a latent hearing which only needs to be awakened[4]; they are chided for being willfully inattentive.[5] Yet every person who finds his hearing failing strives to hold his place in life. It is despair rather than laziness which accounts for his lack of attention.

Those who have lost to any extent their ability to hear are all too well aware of the handicap under which they labor and live. To be viewed by their friends as members of a different race, constantly to receive silly advice as to how their hearing may be restored to normal, increases enormously their difficulties. The public should be educated concerning the causes of hearing defects; it must be told about the aids to hearing which science has devised. When such knowledge is general, those with inadequate hearing will no longer feel isolated from the normal hearing world.

THE BLIND RECEIVE SYMPATHY BUT NOT THE HARD-OF-HEARING

It is strange that those who have hearing defects do not evoke sympathy.[6] Consider for a moment the jokes told about the hard-of-hearing, the cartoons showing the old-fashioned ear trumpet, or the open hand held behind the ear.

Such callousness can only be due to lack of imagination upon the part of the hearing public. No jokes are made about the blind, for there is a more general awareness of what loss of sight could mean. Yet the deaf deserve as much sympathy as the blind for "in them there is not the quickening of all other perceptions and functions."[6]

Helen Keller found lack of hearing a greater loss than her sightless condition. She wrote:

> **"I am just as deaf as I am blind. The problems of deafness are deeper and more complex, if not more important, than those of blindness. Deafness is a much worse misfortune. For it means the loss of the most vital stimulus—the sound of the voice that brings language, sets thought astir, and keeps us in the intellectual company of man."**[7]

Helen Keller is totally deaf. Yet what she wrote applies as well to all whose hearing loss is great enough to make it difficult to communicate easily by sound in a world where most men hear normally. Great as Miss Keller's handicaps were, she overcame them to such an extent that she could live in the hearing world.

All who have any loss of hearing, and especially those whose hearing difficulties are becoming progressively worse should, and must, be encouraged to use any means to adjust themselves to the world rather than to follow their natural inclination to withdraw from it as deafness increases.[8]

1. Best, Harry: Deafness and the Deaf in the United States. The Macmillan Co., New York, 1943, Introduction.
2. Tr. Am. Otol. Soc., Inc. p. 126, 1940. Quoted by Wishart, D. E. S.: Laryngoscope **57**: 444 (July) 1947.
3. Barnett, H. N.: M. Press **216**: 174 (Sept. 4) 1946.
4. Best, Harry: Idem. Chap. 18.
5. Ewing, I. R. and Ewing, A. W. G.: The Handicaps of Deafness. Longmans, Green and Co., New York, 1938. Chap. 1.
6. Barnett, H. N.: M. Press **216**: 84 (July 31) 1946.
7. Keller, H.: Helen Keller in Scotland. Methuen and Company, London, 1933.
8. Ewing, I. R., and Ewing, A. W. G.: Idem. Chap. 2.

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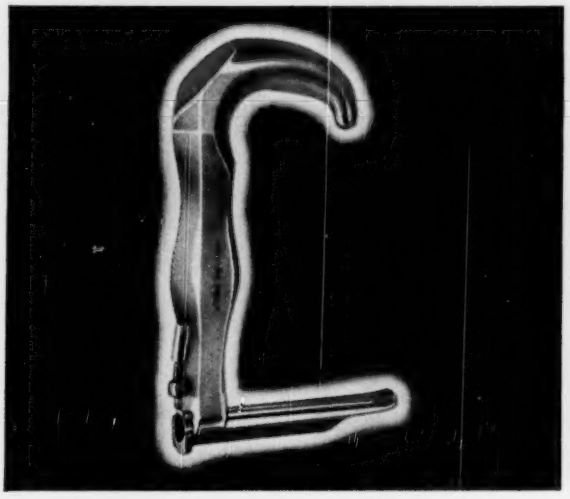

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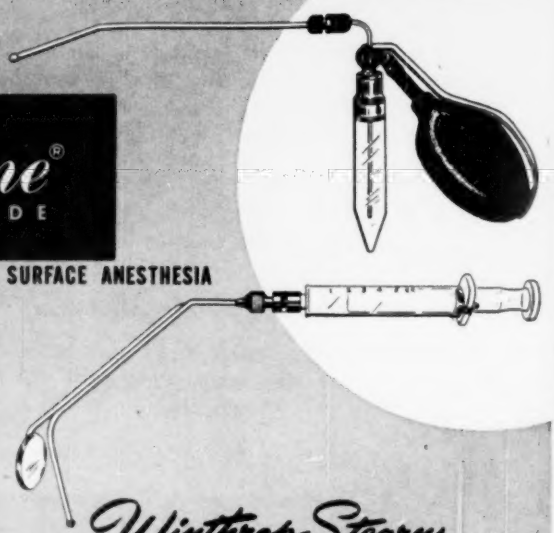

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1. Carabelli, A.: *Diseases of the Chest*, 15:532, May, 1949.
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PLIGHT OF THE NERVE-DEAF PATIENT

The Uselessness of All Present Therapy; the Practical Usefulness of Aural Rehabilitation

L. E. MORRISSETT, M.D.
GREENWICH, CONN.

THIS thesis is the outcome of one otologist's personal experiences with the deafened and hard of hearing in a prewar private practice of six and a half years; a war experience of three and a half years, most of which was spent in intimate contact with casualties of this type in a position of authority in the Army Program for the Aural Rehabilitation for the Deafened and Hard of Hearing, and, finally, a short postwar experience in private practice, continuing to date.

This triple experience has led me to serious reflections on the total inadequacy of present methods of managing deafness, particularly nerve deafness as it occurs in adults in private, and what might be termed nongovernmental, practice. It has also led me to certain conclusions, which, paradoxically, are both specific and vague, about what should be done to improve these methods.

While my particular concern is with nerve deafness, I need scarcely point out that much of what I shall say has to do with deafness of any other origin in the adult. For many years a great deal of useful, valuable and entirely realistic work has been done for deafened children, and they are not considered in this communication.

RECENT LITERATURE ON NERVE DEAFNESS

A review of the recent literature on nerve deafness (recent being interpreted to mean the past fifteen to eighteen years) leaves the thoughtful reader extremely depressed.[1] If he faces the facts honestly he must conclude that not more than a quarter, at most, of all patients with acquired deafness of any considerable degree are amenable to medical

Candidate's thesis presented in partial fulfilment of the requirements for membership in the American Laryngological, Rhinological and Otological Society.

1. Attention is called to the value of the summaries of the bibliographic material on chronic progressive deafness, by George E. Shambaugh Jr.: (*a*) Recent Advances in the Diagnosis and Treatment of Deafness, Ann. Otol., Rhin. & Laryng. **47**:636-648 (Sept.) 1938; (*b*) Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Inner Ear: Summaries of the Bibliographic Material Available in the Field of Otolaryngology, Arch. Otolaryng. **33**:436-474 (March) 1941; (*c*) ibid. **41**:147-154 (Feb.) 1945.

or surgical measures. That is, indeed, a rather liberal estimate. He must also conclude, no matter how much he may wish to be charitable, that the contributions which report new methods of treatment of nerve deafness are extremely unconvincing. The outstanding impressions to be derived from these contributions may be summed up as follows:

1. The background of reported successes in the treatment of nerve deafness is in many instances of doubtful validity, because only successes are reported, failures being minimized or not reported at all, while control series in the true sense of the term are usually completely lacking.

2. In many instances scientific (that is, audiometric) proof as to the supposed improvement does not exist. Not infrequently results are reported as "some improvement," "great improvement," "remarkable improvement," or under similar terms, without the audiometric details which would make evaluation of those results possible by others than the author of the report. It is quite true, as will be pointed out later, that from the standpoint of the patient, his ability to hear conversation is what matters, but from a scientific standpoint this criterion is totally inadequate. Subjective considerations enter too heavily into the whole problem of hearing for therapeutic results to be accepted as valid without complete audiometric substantiation.

3. Even when audiometric details are supplied, criteria of improvement seem inexcusably elastic. Single pretreatment and post-treatment audiograms are not enough to warrant conclusions. It is well known that variations of plus or minus 7 decibels are to be expected. Audiometric improvement must be well above the limits of normal variation to receive serious consideration. The pretreatment auditory loss should be the average of several audiograms taken at intervals of several days, and the post-treatment improvements should be similarly calculated. Jones's [2] important studies on methods for testing auditory function make clear the unreliability of numerous methods which have been used in the past and point to the need for more reliable methods of diagnosis.

4. Furthermore, post-treatment results are not clearly stated in terms of time, and the time of post-treatment observation is often not long enough to permit reasonable conclusions about the permanency of results. In many reports in the literature the duration of the period of post-treatment observation is either not stated at all or is absurdly brief. Improvements in hearing which follow within thirty or sixty minutes or within similarly short intervals after an injection of any substance depend on the patient's word and require careful scrutiny.

2. Jones, M. F.: Discussion of Present Methods for Testing Auditory Function, Ann. Otol., Rhin. & Laryng. **57**:311-323 (June) 1948.

If they occur at all, they are obviously functional. Improvements reported as persisting after weeks or even months are still within the limits in which a psychic lift might be operative with continued treatment. A careful study of available contributions shows that in almost all reported successes the period of observation, if it was stated at all, was a matter of weeks or months. Only occasionally did post-treatment observation cover a longer period, and the longest interval noted between treatment and the termination of the period of observation was practically always considerably less than two years.

5. In the contributions in which audiometric details are stated, a not insignificant number of persons showed actual losses while they were under treatment. It seems fair to assume that these losses represented progressive nerve degeneration, which was completely unaltered by the therapeutic measures employed.

6. In some instances the claimed improvement was entirely on a *post hoc ergo propter hoc* basis, which is generally admitted to be a most unsound kind of evidence. Such improvement, for instance, as followed combined injection of various substances and treatment of blockage of the eustachian tubes seems far more likely to have been the result of the latter method than of the former. Blockage of the tubes can usually be relieved by repeated inflations, and the addition of injections of neostigmine (prostigmin®) or of vitamins would seem to play a highly questionable role in any improvement in hearing which might follow the combined therapy. In fact, it is doubtful in the highest degree that deafness which responds to this type of combined treatment has any significant nerve element in it at all.

7. The preceding comment logically leads to the conclusion that it is highly unlikely that deafnesses of various origins would respond to any one mode of treatment. A patient whose deafness is the result of acute otitis media, for instance, or of so-called secretory otitis can be expected to improve under standard measures of treatment. In fact, he can be expected to improve spontaneously, and frequently does. The plight of the patient with nerve deafness is entirely different. Statistical and clinical reports of therapeutic methods to which all varieties of deafness respond must obviously carry very little weight. En masse reports are unconvincing, and only those results can be accepted in which cases of deafness are classified etiologically.

8. It is true, as Howard Kelly once wisely remarked in another connection, that what the patient is looking for is a cure, with or without a rational basis behind it; but the fact remains that when specific remedies are used for nerve deafness, it is not unreasonable to require that some connection be evident between the basic pathologic condition and

the therapeutic method. The strongest argument, according to Shambaugh,[1b] against the causative role of vitamins in nerve deafness is the fact that loss of hearing is no part of the clinical picture in scurvy, beriberi, pellagra and other vitamin deficiency diseases. If nerve deafness is not associated with such clearcut vitamin deficiencies as these, it is difficult to accept the thesis that it may be caused by a subclinical avitaminosis.

9. While a great deal of commendable experimental work has been done to demonstrate the effect of vitamin deficiencies on the auditory apparatus, particularly on the nerve elements,[3] the results in animals and their applicability to human subjects must be evaluated with a great deal more caution than has sometimes been displayed. Lurie[4] has summarized the situation very well: It has been possible experimentally to produce lesions of the sense organ and the conduction apparatus and to demonstrate microscopically the pathologic changes which have occurred, but it has not been possible to state, even approximately, how much hearing has been lost as the result of these experiments. Moreover, while guarded experiments of various sorts in the human subject have brought about transitory hearing losses (some of which have been of alarmingly long duration), it has not been possible to demonstrate what has happened, or what may happen, to the human sense organ as a result.

Lurie also commented on the fact that most experimental work which has been successful from the therapeutic standpoint has been done with young and rapidly growing animals; when adult animals were used, the results were less conclusive. Those who desire to translate results of animal experiments to human subjects obviously must bear in mind that nerve deafness in the human subject is usually a disorder of advancing years.

Finally, Lurie called attention to the fact that animal experimentation apparently indicates that nerve deafness is primarily the result of changes in the organ of Corti and that these changes, once a certain point is reached, are irreversible. Here animal observations are extremely suggestive: In the human subject the evidence seems to indicate that when nerve deafness is unmistakably manifest, irreversible changes have already occurred in the end organ or the auditory nerve.

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3. Covell, W. P., and Noble, L.: The Significance of Myelin Sheath Degeneration for the Cochlear Nerve, Ann. Otol., Rhin. & Laryng. **46**:895-911 (Dec.) 1937; Pathologic Changes in the Peripheral Auditory Mechanism Due to Avitaminoses (A, B complex, C, D and E), Laryngoscope **50**:632-647 (July) 1940.

4. Lurie, M. H.: Medical Progress, Otology; The Treatment of Deafness in the Light of Recent Animal Experimentation, New England J. Med. **226**:886-890 (May 28) 1942.

10. The literature is additionally confused by the fact that numerous reports deal with tinnitus in association with deafness, while other series include cases of deafness and of tinnitus. In many instances patients reported disappearance of, or improvement in, tinnitus following treatment, and in some cases hearing also seemed to have improved. For a number of reasons it would be a mistake to read very much into an improvement in hearing associated with an improvement in tinnitus. The essential nature of tinnitus, the emotional component frequently present,[5] and the impossibility of determining by objective means the original degree of annoyance and the post-treatment degree of relief make it impossible to evaluate in any scientific manner the possible results of therapy. There is no doubt, however, that a patient whose tinnitus has improved—or, more correctly, annoys him less—is likely to hear better if only because he has one less distraction to contend with. If there is actual proof of an improvement in hearing, the masking effects of tinnitus may provide the explanation; tinnitus of a certain vibration might mask the sounds of a certain frequency; with improvement in tinnitus, sounds of that frequency would again be audible.

11. Finally, a critical review of the recent literature, such as I have just conducted, makes clear one extremely significant fact: Almost every report on new therapeutic measures for nerve deafness is entitled "Preliminary Report." This is eminently proper. But, with a few exceptions, one looks in vain for subsequent reports by the original authors, while others who test the methods described in general fail to confirm the original good results.

SAMPLE DATA

A complete review of the extensive literature surveyed in the preparation of this thesis would not be profitable, but certain illustrative data will be briefly summarized:

Neostigmine Therapy.—Davis and Rommel,[6] in 1939, seem to have been the first to suggest the use of neostigmine in nerve deafness. Rommel, who was using the drug in the treatment of myasthenia gravis, gave himself an injection of it to test its effect on the tissues; when he observed improved auditory acuity in himself, he applied the method to deafened patients. It is difficult to determine from the authors' report exactly how many patients were treated and exactly what types of deafness they presented. It is clear, however, that a large number suffered from acute blockage of the eustachian tubes "with tinnitus aurium and

5. Fowler, E. P.: The Emotional Factor in Tinnitus Aurium, Laryngoscope **58**:145-154 (Feb.) 1948.

6. Davis, T. C., and Rommel, J. C.: Treatment of Deafness and Contiguous Nervous Disorders with Prostigmine: A Preliminary Clinical Report, Arch. Otolaryng. **29**:751-758 (May) 1939.

usually marked deafness." Twenty-four cases in which improvement occurred were recorded in some detail, but the period of observation was not evident and pretreatment audiometric studies were not carried out or were not mentioned.

The good results reported by Davis and Rommel with neostigmine have not been duplicated by others. Ersner and his associates,[7] for instance, treated 59 patients by this method according to various dosage schedules; all were tested with calibrated audiometers. Four patients said that they heard more clearly but there was no audiometric confirmation of the improvement, and similarly good results were secured with injections of sodium chloride. Eighteen patients showed transitory audiometric improvement ranging from 8 to 10 decibels, but the results were not regarded as impressive in view of the normal variations in audiometry already commented on.[2] Treatment had to be discontinued for 3 patients because of anginal attacks, and the remaining patients showed no improvement at all.

Schluederberg,[8] who treated 38 patients with neostigmine, reported improvement with 5 patients, in all of whom tinnitus was also present. He called attention, however, to the carefully controlled study of 56 similarly treated patients, carried out by Houser, Campbell and himself,[9] in which deafness was not improved in a single instance, although 7 patients thought they had experienced some lessening of tinnitus. Van Loon and Seltzer[10] used neostigmine in 22 cases but observed no improvement in deafness in any case in which there was not also some improvement in tinnitus. Alexander,[11] who stated the opinion that neostigmine was of benefit in deafness due to otitis media, in combination with other methods of treatment, found it useless in nerve deafness. His experience may be taken as typical.

Vitamin Therapy.—An ample literature is concerned with the treatment of nerve deafness by vitamins, although controlled series of cases are few. Veasey,[12] in 1940, stated that vitamin B therapy might help

7. Ersner, M. S.; Rush, I. A., and Myers, D.: Prostigmine Methylsulfate in the Treatment of Deafness and Tinnitus Aurium: Report of Cases with Negative Results, Arch. Otolaryng. **33**:193-198 (Feb.) 1941.

8. Schluederberg, H.: The Treatment of Deafness and Tinnitus Aurium with Prostigmine, Ann. Otol., Rhin. & Laryng. **51**:526-530 (June) 1942.

9. Houser, K. M.; Campbell, E. H., and Schluederberg, H.: The Treatment of Chronic Deafness and Tinnitus Aurium with Prostigmine, J. A. M. A. **115**:994-995 (Sept. 21) 1940.

10. Van Loon, E. L., and Seltzer, B. P.: The Use of Prostigmine in Deafness and Tinnitus Aurium, M. Woman's J. **48**:352-354 (Nov.) 1941.

11. Alexander, L. W.: The Use of Prostigmin Methylsulfate in Deafness: A Preliminary Report, New Orleans M. & S. J. **93**:474-476 (March) 1941.

12. Veasey, C. A., Jr.: Nature of Vitamin B and Its Components, with Special Reference to Nerve Deafness, Arch. Otolaryng. **31**:74-93 (Jan.) 1940.

some patients with nerve deafness, though it was impossible to select them on any evident basis, but the 6 cases which he reported do not seem to substantiate his contention. Babbitt [13] mentioned that he treated 100 patients with nerve deafness with vitamin B and iron over a measured period, with almost immediate, and sometimes dramatic, response to the initial administration in some cases but with no permanent improvement. His explanation was that the treatment supplied a neurophysiologic stimulus which required further nutritional support for its continuance.

Childrey [14] treated 26 patients with nerve deafness with adequate doses of vitamin B over prolonged periods of time (2 to 46 weeks, an average of 7.3 weeks) and supplemented the treatment with vitamin-rich foods. The audiograms showed no improvement in auditory acuity in any instance, and the hearing of some patients became worse under treatment.

Shambaugh and Jennes,[15] after a previous unsuccessful experience with smaller doses, treated 6 patients with nerve deafness with intravenous injections of 16,650 international (U. S. P.) units of thiamine hydrochloride six times a week for two weeks and observed no improvement in any instance. Their own explanation of the good results sometimes reported from this method of treatment is that patients whose hearing is improved may be suffering from secretory otitis media, either alone or in association with nerve deafness.

Selfridge,[16] an exponent of vitamin therapy for nerve deafness, expressed the belief that if hopeless damage to the eighth nerve has not already occurred (a hearing loss in the frequencies between 240 and 4096, in the voice range) vitamin B therapy may be helpful, but as yet there is no scientific proof for his theory.

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13. Babbitt, J. A.: A Study of Medical and Surgical Aids to Hearing, Surg., Gynec. & Obst. **68**:395-400 (Feb. 15) 1939.

14. Childrey, J. H.: Vitamin B and Other Measures in the Treatment of Deafness, Laryngoscope **50**:648-657 (July) 1940.

15. Shambaugh, G. E., Jr., and Jennes, M. L.: Therapy of Nerve Deafness and Tinnitus Aurium: Use of Large Doses of Thiamine Hydrochloride and Evaluation of Results with a Source of Possible Error in Interpretation of Improvement, Arch. Otolaryng. **35**:513-522 (April) 1942.

16. Selfridge, G.: Can Certain Diseases of the Ear, Nose and Throat, Especially Degeneration of the Eighth Nerve, Be Classified as "Deficiency Diseases"? Laryngoscope **46**:85-106 (Feb.) 1936; Chronic Progressive Deafness from a Nutritional Standpoint: A Preliminary Report, Ann. Otol., Rhin. & Laryng. **46**:865-894 (Dec.) 1937; Nicotinic Acid and the Eighth Nerve, ibid. **48**:39-53 (March) 1939; The Eighth Nerve in Relation to Thiamin Chloride and Nicotinic Acid, ibid. **48**:419-432 (June) 1939; Eighth Nerve Deafness from a Nutritional Standpoint: A Further Contribution, ibid. **48**:608-631 (Sept.) 1939.

Benzyl Cinnamate Therapy.—By 1946 Jacobson,[17] over a three and a half year period, had treated 240 patients with benzyl cinnamate by intramuscular injection. Of one hundred and eighty-five questionnaires sent out to these patients, one hundred and thirty were returned. Sixty-one per cent of the group which replied reported improved hearing, and 82 per cent reported diminution in tinnitus or its complete disappearance. Although the author mentioned audiometric testing, he supplied no details. The best results were achieved in chronic inflammatory lesions of the middle ear, but otosclerotic and nerve deafness was also improved in a "limited number" of cases. Jacobson's solution does not seem to have had any general popularity in the treatment of any variety of deafness.

Amino Acid and Vitamin Therapy.—In 1946 Hirschfeld, Jacobson and Jellinek[18] reported the treatment of deafened patients with a combination of amino acids and vitamins. Jacobson had used this method for gastrointestinal disorders, and its use had been extended to deafened patients because of the improvement in hearing reported by a deafened subject who had been thus treated. Details were not supplied, but it is stated that increases in hearing were observed in some cases in from fifteen to thirty minutes after the intramuscular injection, that the full effect was usually reached in from two to four weeks and that some patients had maintained their gains. "Permanent cure" is mentioned, but the detailed tables give no dates, and the audiograms included in the paper for the most part cover limited periods of time. The authors expressed the belief that their results indicate either that the conditions of the organism affecting hearing have been permanently improved by the treatment employed or that the auditory nerve has undergone permanent changes of a "restitutive" nature. A more reasonable explanation seems to lie in their observation that patients treated by this method showed increased resistance to fatigue, which is known to play an important role in loss of auditory acuity. This method does not seem to have been widely employed.

The Treatment of Psychogenic (Functional) Deafness by Narcosynthesis.—Although nerve deafness is on an organic, not a functional, basis, the Army experience in World War II showed clearly that the organic basis may have a functional overlay. I have frequently observed this

17. Jacobson, J.: Treatment of Certain Forms of Deafness by Means of Benzyl Cinnamate: A Preliminary Report, Arch. Otolaryng. **38**:365-368 (Oct.) 1943; Treatment of Certain Forms of Deafness, Tinnitus and Dizziness with Benzyl Cinnamate, Eye, Ear, Nose & Throat Monthly **23**:396-399 (Oct.) 1944; Roentgenographic Changes in the Mucosa of the Mastoid Cells Following Treatment of Deafness with Benzyl Cinnamate, ibid. **25**:547-553 (Nov.) 1946.

18. Hirschfeld, H.; Jacobson, M., and Jellinek, A.: New Treatment of Hearing Disorders, Arch. Otolaryng. **44**:686-700 (Dec.) 1946.

association in civilian patients I have seen since my return to private practice. This group of patients, again as the Army experience showed, can be helped by narcosynthesis. Furthermore, the mass trial of this method in the Hoff General Hospital Aural Rehabilitation Center during World War II established the fact that in properly selected cases this method is useful whether the deafness is of recent or remote origin.[19]

Approximately 15 per cent of the 1,375 deafened and hard of hearing casualties treated at the Hoff installation were found to have an entirely functional basis for their deafness or to have a functional overlay on an organic basis. One hundred and two patients with this type of deafness were treated by narcosynthesis over the nine month period ending October 1945, three fourths of whom showed a complete return of hearing or a great improvement in auditory acuity, both clinically and audiometrically. For 42 of these patients the defective hearing antedated induction, and for 25 patients it had lasted for more than ten years. These figures suggest the value of narcosynthesis in selected cases of nerve deafness. On the other hand, the number of patients with this type of deafness who can expect to be benefited by this method is small and its contribution to the major problem is equally small.

The Fenestration Operation.—The fenestration operation, which is now generally performed with the nov-ovalis technic devised by Lempert,[20] represents by far the greatest advance ever made in the therapy of deafness. It is directly applicable, however, only to deafness due to clinical otosclerosis and is definitely contraindicated when nerve deafness is associated. At the present time, as Rosenwasser[21] has pointed out, no accurate, infallible test has been developed for the estimation of reserve cochlear nerve function in clinical otosclerosis to aid one in making a decision as to the suitability of the operation in borderline cases. When cochlear damage is clearcut, however, there is no justification for operation.[22]

Personal Experiences.—I might conclude this depressing recital with a brief mention of my own lack of results with the various methods of treatment suggested for nerve deafness. Like all conscientious otologists,

19. Bergman, M.: Narcosynthesis in the Management of Functional Deafness, to be published.

20. Lempert, J.: Fenestra Nov-Ovalis: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **34**:880-912 (Nov.) 1941; Lempert Fenestra Nov-Ovalis Operation for the Restoration of Serviceable Unaided Hearing in Patients with Clinical Otosclerosis: Its Present Evolutionary Status, ibid. **46**:478-511 (Oct.) 1947.

21. Rosenwasser, H.: When Is a Case of Clinical Otosclerosis Suitable for Fenestration? Laryngoscope **58**:244-253 (March) 1948.

22. Maxwell, J. H.: Training of the Surgeon and Selection of Patient for the Fenestration Operation, Arch. Otolaryng. **46**:539-543 (Oct.) 1947.

I have been troubled by my inability to help most deafened patients. In these circumstances, one grasps at straws and tests every new method suggested, however irrational it may seem, so long as it does no harm.

In round numbers, I have treated 40 deafened patients with neostigmine, 25 with Jacobson's solution (benzyl cinnamate), 50 with vitamin B and 35 with antihistaminic drugs (diphenhydramine [benadryl®]; a chemical combination of histamine and despeciated horse serum globulin, containing no free histamine [hapamine®]; diphenhydramine aminophylline [hydrillin®]; tripelennamine [pyribenzamine®], and a preparation from desiccated kidney and extract of intestinal mucosa of hogs, containing histaminase [torantil®]). In addition, I have operated on the nasal septum in over 100 deafened patients and have inflated the eustachian tubes and operated on the sinuses, the pharynx and the nasopharynx in several hundred others.

The patients in the surgical groups needed the surgical procedures instituted, without regard for their deafness, which was either not improved at all or was improved to a negligible degree by the operation. The patients in the other groups sometimes showed transient clinical improvement—which even apparently hopelessly deafened patients seem to show when a new treatment is instituted—and some disappeared from observation while they were still in this stage of improvement. I take no comfort in the thought that this improvement perhaps continued. I feel reasonably sure that it did not. When the period of observation and follow-up was adequate, not a single patient with nerve deafness whom I have ever treated, regardless of the method of treatment or the duration of treatment, showed any significant sustained improvement.

Tinnitus furnished something of an exception to my generally poor results in that it was sometimes improved and occasionally disappeared altogether with certain methods of treatment. Tinnitus was present in the majority of patients treated by intramuscular injections of neostigmine and was apparently helped in a few cases in that the pitch was altered. Results with vitamin therapy were substantially similar. Tinnitus was never improved when benzyl cinnamate or the antihistaminic drugs were employed. The deafened patient's lot is naturally improved if tinnitus is decreased or eliminated, but, as already pointed out, too many subjective factors are associated with this symptom to permit a scientific evaluation of results of special therapeutic measures.

My own experience with the treatment of nerve deafness strongly suggests that if the successes reported in the literature as following therapy with vitamins, neostigmine and other agents were properly followed up and were audiometrically controlled, either all improvement would prove to be transient or such improvement as seemed at all permanent could be explained in three possible ways: 1. It was psychologic,

this type of improvement sometimes being sustained to some degree over a varying length of time. 2. The deafness was on a psychogenic basis. 3. The patient's general condition was so improved that his alertness was increased and his auditory acuity correspondingly improved. Fatigue and lassitude have a proved deleterious effect on the sense of hearing, and vitamin therapy and other tonic therapy are always justified in an attempt to build up the general health.

AURAL REHABILITATION, THE PRESENT SOLUTION OF THE PROBLEM OF NERVE DEAFNESS

The present situation in respect to the management of pure nerve deafness may be summarized about as follows: 1. No medical measures have proved to be of any permanent value. 2. No direct surgical attack on the underlying pathologic process is feasible. 3. Narcosynthesis is useful in a small number of cases in which a functional overlay increases the disability. 4. The fenestration operation devised for the relief of deafness of otosclerotic origin is perhaps useful in borderline cases in preventing further nerve degeneration, but the number of such cases is small at best and no satisfactory tests for their selection now exist.

Since direct medical and surgical measures are useless in nerve deafness, and since narcosynthesis and the fenestration operation are only indirectly useful and are applicable to only a very small proportion of cases, what help is there for the great body of patients with nerve deafness?

This group of patients represents a special problem, the magnitude of which is as yet entirely unknown. In fact, the magnitude of the whole problem of deafness in the United States is unknown. Estimates vary from as low as 6,000,000 to as high as 15,000,000 persons, these figures applying, of course, to persons with all types of deafness. The most scientific and the best controlled study made to date is the survey carried out in 1935 and 1936, as part of the National Health Survey.[23] This survey revealed that for the population sampled (2,502,391 persons, 48 per cent of whom were men), 1 out of every 78 men and 1 out of every 85 women had a hearing handicap. Between the ages of 35 and 44 years, 1 out of every 103 men and 1 of every 108 women had a handicap from deafness, the incidence rising with each succeeding decade until between the ages of 65 and 74 the distribution was 1 out of every 14 men and 1 out of every 18 women. An interesting and significant feature of the survey was the greater concentration of hard of hearing persons in the adult years in the lower income and the unemployed

23. Beasley, W. C.: The General Problem of Deafness in the Population, Laryngoscope **50**:856-905 (Sept.) 1940.

groups. Since nerve deafness accounts for a large proportion of adult deafness, it can be assumed to be an occupational as well as a personal and social liability.

To date, we otologists have not done a great deal toward solving the problem of nerve deafness, chiefly because we have not realistically faced the fact that this type of deafness, in the light of present knowledge, is incurable. What most of us have done in the past was (1) to give the patient the addresses of retailers selling hearing aids, (2) to urge him to cooperate with lay agencies and, in particular, (3) to become a member of the local League for the Hard of Hearing, in which he could secure lessons in lip reading, investigate vocational opportunities and make social contacts with similarly handicapped persons. These methods in some ways solve the problem. But there is general agreement, at least in theory, that the selection and fitting of hearing aids should be a medical, not a commercial, matter, and by handing over the management of deafened persons to lay organizations, however admirable their work may be, we are by-passing a responsibility, which should not be done unless these lay agencies fitting hearing aids are managed by physicians.

About twenty years before World War II, the late Charles H. Mayo made the prophecy that within the next two decades rehabilitation would come to be the master word in medicine. That prophecy is rapidly coming true, its fulfilment having been greatly expedited by the events of World War II. In the opinion of many observers, the Army Aural Rehabilitation Program for the Deafened and Hard of Hearing advanced the care of deafness by at least twenty years. Even before the outbreak of the late war there was a growing realization, frequently translated into action, that while the physician's immediate and urgent task was to save his patients from death and enable them to continue to live, his ultimate responsibility was their restoration to complete economic usefulness and to a normal social life, or, if those objectives were impossible, to as close an approximation of normal status as could be achieved.

That accomplishment is the solution of the problem and should be the objective of the otologists called on to handle the problem of nerve deafness. It is a perfectly practical solution. Our greatest failure as otologists is that we have not realized—or, if we have realized it, we have not acted on the realization—that a deafened patient who is not adjusted to the life he must live represents a failure of treatment. This realization is well expressed by the motto of the Reading (Pennsylvania) Hospital rehabilitation clinic for the aurally handicapped: "Any deaf or hard of hearing person may represent a phenomenal surgical or medical success, but if he is not equipped to fit readily into the pattern of his civilian life he will represent a failure as far as vocational rehabilitation

is concerned."[24] There has been, as yet, no "phenomenal surgical or medical success" in any nerve-deafened patient, but there is no reason why, in the future, there should continue to be so large a proportion of failures in their adjustment to life. In aural rehabilitation, and in aural rehabilitation alone, lies the present salvation of the nerve-deaf adult, and otologists would do well to face that fact realistically and promptly.

A realistic facing of that problem immediately brings up other problems. In addition to the points already listed as derived from recent articles dealing with methods of treating nerve deafness, another consideration must impress the thoughtful reader of the recent literature: It contains a great deal about aural rehabilitation, but the articles on the subject fall into two groups, (1) those dealing with general principles of management and (2) those dealing with aural rehabilitation in the Army, Navy and Veterans Administration hospitals and, occasionally, in a public or, very occasionally, in a private hospital. There is no provision, in other words, for the great body of deafened adults who are not eligible for federal assistance.

Let us follow the reasoning to its logical conclusion.

1. If the point of view be accepted—and I personally believe that no other is warranted—that nerve deafness is generally irreversible and that the patient's only chance of readjustment to life lies in aural rehabilitation in the full sense of that term, otologists must move rapidly to translate theory into practice.

2. At the present time there are ample facilities for the aural rehabilitation of Army, Navy, Air Force and Veterans personnel, though perhaps more effort should be devoted to educating veterans on the acceptance and full use of these facilities.

3. Most public hospitals, practically all private hospitals, including specialty hospitals, and most otologists in private practice are not equipped to handle deafened and hard of hearing patients as they can be handled in federal facilities.

4. Unless otologists in private practice take steps to remedy this situation, deafened and hard of hearing patients, once the possibilities of aural rehabilitation become generally comprehended, will inevitably demand that facilities for this purpose be provided for them. If these facilities are not available privately, they will demand them from the government, and it is unreasonable to expect that they should follow any other course.

5. Otologists, who presumably are as eager as other physicians to prevent further encroachment by federal agencies on the field of private practice, would do well to reflect seriously on the latter possibility.

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24. Walter, M. M.: Vocational Rehabilitation for the Deaf and Hard of Hearing, Tr. Am. Acad. Ophth. **49**:103-111 (Nov.-Dec.) 1944.

The needs of the deafened adult place the otologist on the horns of a dilemma. He naturally desires to supply those needs. There is, in fact, no good reason why the benefits provided for Army and Navy casualties (many of them, it must be remembered, had their hearing disability before they entered the service) and now provided for veterans should not be provided for others. Yet they are not. So far as hearing disability is concerned, the deafened adult not eligible for federal benefits is not provided for at all, except in occasional public hospitals, where hearing clinics have been set up. The deafened adult who is a private patient is for all practical purposes the forgotten man.

What are the needs of the nerve-deaf adult? At a minimum they are essentially the needs presented by the deafened casualties of World War II. He must have (1) complete medical and otologic study; (2) audiometric tests, tests of auricular ability and similar tests; (3) psychologic study if it seems indicated; (4) provision of a hearing aid; (5) instruction in lip reading; (6) auditory training; (7) voice training and speech correction if they are indicated; (8) speech insurance if it is indicated, and (9) vocational guidance if it is indicated. In other words, the deafened private patient needs precisely the same sort of basic program as was provided for the Army casualty, though it must be applied with an elasticity which the Army Program, even though it endeavored to individualize each patient, could not possibly achieve. The civilian patient, on the other hand, does not ordinarily present many of the problems inherent in the fact that the deafened Army casualty was in service and necessarily regimented. In particular, the private patient frequently, indeed usually, comes to the otologist from the midst of his routine occupations and he can continue to lead his usual normal life while he is under treatment. This gives him an enormous advantage over the deafened Army casualty, for it means that he does not suffer from the "disintegrating effect" of idleness.

How practical is such a program in private otologic practice? The question must be answered from three points of view, concerned, respectively, with equipment, personnel and economics, though actually economics underlies equipment and personnel also.

The equipment provided in Army, Navy and Veterans aural rehabilitation centers is undoubtedly more than is needed for ordinary civilian rehabilitation centers, aside from the fact that it was purchased under the exigencies of war. On the other hand, the activities of an aural rehabilitation program require space and require special equipment, particularly electronic equipment, for which there is no acceptable substitute. There has been a good deal of argument as to whether or not a soundproof room [25] is needed on the ground that normal speech

25. Soontup, T. L., and Bergman, M.: Soundproof Construction for Aural Rehabilitation, Prog. Architect., March 1948, pp. 70-75.

and normal hearing are conducted against a background of environmental noise. The argument, of course, is specious. A sound-treated room, at the least, is necessary for accurate testing, just as a certain minimum of equipment is desirable, including audiometer, turntable and playback unit, amplifier, loudspeakers with good fidelity (one to transmit low frequencies and another, or several, to transmit high frequencies faithfully); headphones, an additional turntable, amplifier and loudspeaker combination, together with several dozen recordings, for auditory training, and a recording machine for speech correction. A magnetic tape recorder is desirable, if the budget permits, and a clinic for the acoustically handicapped should have several desk amplifiers to facilitate the initial interviews.[26] Even if the sound-treated (rather than soundproofed) room and the equipment are provided with all possible economy, the initial expenditure is large enough to make the average private physician hesitate before embarking on it.

Equipment, moreover, is only part of the expenditure. There is next the question of personnel. Obviously the otologist cannot single-handed carry out the program which has been outlined, even if he were himself qualified to do so, which in most instances he is not. Lip reading is a profession in itself. Speech correction is another specialty. Psychologic investigation may not be needed in every case, but it requires the services of a specialist when it is necessary. Narcosynthesis is applicable in only the occasional case of nerve deafness in which there is a functional overlay, but it can be conducted only in cooperation with a psychiatrist and it is a time-consuming form of treatment. In other words, in addition to providing expensive equipment, the otologist who undertakes an aural rehabilitation program must also provide highly trained personnel to handle the equipment and to instruct the patient in the things he must learn in the course of his adjustment to his new life.

These two necessities lead to a consideration of the third problem in aural rehabilitation, the financial problem. Will the private patient pay for the expensive set-up required for his rehabilitation? The answer is obviously no, he will not. Such treatment would be prohibitively expensive and would be quite beyond the reach of the average patient.

What, then, is to be done to provide aural rehabilitation for the private patient? The answer is simple—the provision of mass facilities. Ideally, certain hospitals in each community, particularly specialty hospitals, should and could provide them. If they cannot or will not, the

26. This list of basic equipment was provided by (former) Lieut. Moe Bergman, who played an active part in the Aural Rehabilitation Program at Hoff General Hospital during World War II and who is now connected with the Aural Rehabilitation Program in the Veterans Branch Office, New York city.

time is rapidly approaching when the otologists of the community must themselves provide them.

This will be quite a reversal of tradition and practice for such individualists as American otologists are. It will be, in effect, group practice limited to a single field. It will involve combined expenditures, combined supervision of facilities and personnel, surrender of individual patients and surrender of rights and privileges which are a part of private practice but which cannot be maintained in group practice. Legal, financial and professional details must be worked out, but with good will, and with all decisions made in the light of the patient's best interests, there is no reason why difficulties should not be promptly overcome. Once the first step is taken, once this new concept of otologic practice is accepted, other difficulties, I believe, would promptly become minor.

The difficulties have, in fact, been overcome in the few hearing clinics now in operation in the United States. The Otological Research Laboratory at the Abington Memorial Hospital in Philadelphia, under the direction of the late Dr. Walter Hughson, was for many years a brilliant illustration of the admirable work which can be accomplished under the proper setup. The aural rehabilitation center at the Reading Hospital has already been mentioned.[26] A Hearing and Speech Center has been in operation for the past year at the Johns Hopkins Hospital, under the direction of Dr. William G. Hardy. It is an integral part of the department of otolaryngology and is a university as well as a hospital unit. It receives patients directly or on referral, and private, semiprivate and dispensary patients are treated. Fitting of hearing aids is the first step in hearing rehabilitation, and "communicative reeducation" the second.[27]

Another hearing clinic has recently been established at the Eye, Ear, Nose and Throat Hospital in New Orleans, under the direction of Dr. Lucian W. Alexander. It will treat all varieties of deafness, and management of nerve deafness will be on the basis set forth in this thesis, that the only hope for the patient with this type of deafness lies in his complete aural rehabilitation.[28]

PRACTICAL CONSIDERATIONS OF AN AURAL REHABILITATION PROGRAM

The Army aural rehabilitation program is well known in its essentials and need not be set forth here.[29] Certain practical considerations concerning it might, however, profitably be mentioned.

27. Hardy, W. G.: Personal communication to the author.

28. Alexander, L. W.: Personal communication to the author.

29. Morrissett, L. E.: The Aural Rehabilitation Program of the United States Army for the Deaf and Hard of Hearing, Ann. Otol., Rhin. & Laryng. **55**:821-838 (Dec.) 1946.

Hearing Aids.—Hearing aids have not yet anywhere reached nearly their full usefulness. According to Berry,[30] approximately 800,000 are now in use, which is a small number for a deafened population estimated to number several million. In 1943, again according to Berry, 110,000 aids were purchased; in 1946 the annual purchases had risen to 225,000. While young persons, and even children, may use an aid, the greatest usefulness of this device is among the adult population, the average use, Berry estimates, being at 55 years of age. When one realizes that there are in this country at this time 31,000,000 persons over 50 years of age, it is evident that the present use of hearing aids is limited indeed.

There are a number of explanations for this situation. Generally speaking, a person with a hearing loss of 20 to 45 decibels need scarcely go to the trouble and expense of securing a hearing aid, though his hearing will be greatly benefited by training in residual hearing and in the attention factor. A person with a hearing loss of more than 70 decibels will find little benefit from an aid, and his attention should be chiefly directed to learning lip reading. But the average nerve-deaf adult with a loss of 40 to 70 decibels will be greatly benefited by the use of an aid, if only he can be persuaded to use one and is taught how to use one and is properly adjusted to it. According to Day,[31] 75 per cent of all hearing aids are discarded promptly. Whether or not this figure is correct for the general deaf population it is impossible to say, but it is certainly true that a great many deafened patients who should use hearing aids do not procure them, while others who procure them abandon them promptly.

One simple and practical method which otologists might employ to increase the use of hearing aids by those who need them is to discontinue at once the false comparison of hearing aids and eyeglasses. Actually there is no resemblance between the two devices. In the first place, glasses have been employed for many years, whereas hearing aids are a relatively recent development. In the second place, no hearing test has yet been devised which equals in accuracy the $x/20$ (Snellen) test used by ophthalmologists for many years, and fitting is in large part on a trial and error basis. In the third place, a hearing aid does not bring back hearing as a lens brings back sight, and it is misleading to let the patient believe that it does. Finally, the eye has an accommodative mechanism which permits rapid accommodation to sudden extremes of

30. Berry, G.: Hearing Aids: The Evaluation and Practicability of Hearing Aids, with Review of Recent Literature, Ann. Otol., Rhin. & Laryng. **57**:500-508 (June) 1948.

31. Day, K. M.: Prescribing a Hearing Aid, Pennsylvania M. J. **43**:1299-1302 (June) 1940.

light and darkness. There is no such mechanism in the ear, which does not adjust itself well to the sudden amplification of sound produced by the application of a hearing aid over a long-deafened ear.

Hughson and Thompson,[32] with a proper (but unusual) appreciation of the patient's own reaction to his aid, secured information concerning this reaction from 97 patients, 42 of whom expressed satisfaction, without reservations, as to the service they were receiving from them, while 27 others liked them with reservations. Most of the patients who expressed unqualified satisfaction were using their aids steadily during their waking hours, thus proving clearly the benefits which come from constant use of the prosthesis. Many of the 14 patients who found the aids unsatisfactory were known to be nervous, self-conscious, and highly emotional. It is of interest, in view of the emphasis placed on the improvement of tinnitus by authors who have suggested methods of treatment for nerve deafness, that this symptom was not a leading factor in the results reported by Hughson and Thompson.

Selection and Fitting: This is not the place to discuss the details of the selection and fitting of hearing aids, but it might be pointed out that an aid, to be of any value, must furnish a guaranteed measured gain in speech reception of at least 30 decibels. Modern vacuum tube aids are vastly superior to the older carbon models, but, generally speaking, there is no clearcut superiority between different makes, though an individual patient may receive more help from one type than from another. Work done at the Electro-Acoustic and Psycho-Acoustic laboratories of Harvard University [33] during the war established the fact that most patients, regardless of the type of hearing defect from which they suffer, hear better with an aid which amplifies all frequencies uniformly or one which emphasizes the higher frequencies moderately. The latter type of aid is more likely to be useful to the nerve-deaf patient, whose hearing loss tends to be in the higher frequencies.

Over the past few years, as Berry [31] has recently pointed out, enormous advances have been made in hearing aids. The criterion of hearing improvement has been raised from a minimum of 30 to 45 decibels, and gains up to 95 decibels seem possible in certain circumstances. The cases are stronger, more convenient and better looking. The friction of clothing has almost ceased to be a problem. New batteries are more efficient and are longer lived. The printed circuit recently adopted by certain companies, which saves a large amount of skilled labor in the assembly of small parts and makes for simplicity,

32. Hughson, W., and Thompson, E.: The Hearing Aid from the Patient's Point of View, Arch. Otolaryng. **38**:252-260 (Sept.) 1943.

33. Davis, H.; Hudgins, C. V.; Marquis, R. J.; Nichols, R. H., Jr.; Peterson, G. E.; Ross, D. A., and Stevens, S. S.: The Selection of Hearing Aids (pt. 2), Laryngoscope **56**:135-163 (April) 1946.

strength and protection against moisture, will probably soon come into general use. Finally, prices have been reduced, though they are, in most instances, still too high.

But when all of these advancements are granted, the fact remains, and it is no discredit to the commercial agencies which make the instruments, that hearing aids are manufactured and sold primarily for the profit motive. The manufacturers are in competition with each other, and part of the cost of hearing aids reflects the cost of that competition and of the high pressure advertising which it engenders.

Otologists with a wide experience in the management of deafened patients have taken the position that the selection and fitting of hearing aids should be conducted under medical supervision, their position being no reflection on either the good faith or the competence of the companies which make the aids. The manufacturers, they insist, should be engaged solely in turning out as good aids as possible and at as low a price as possible. It should be the business of the otologist to select and fit the appropriate aid for the individual person, or at least to supervise its selection and fitting. I cannot agree with Macfarlan [34] that the otologist is merely an interested third party, who can be assured that the retailer will give the patient the most suitable instrument for his special needs.

If, however, the otologist reserves to himself the responsibility and prerogatives for selecting and fitting hearing aids, he must assume more than a nominal responsibility for this task. He need not be an acoustic or an electrical expert, but he must acquaint himself with the general mechanism of the various aids, he must know what acoustic amplification can accomplish and he must know how to apply theoretic physical facts to the individual clinical needs of his patients.

Again, this is a major task, expensive, time consuming and impractical for any single otologist. To take care of one patient, he would have to neglect many others. It is therefore one more practical reason for the management of deafened persons in aural rehabilitation centers.

Training in Their Use: Systematic training in the use of hearing aids is an essential part of an aural rehabilitation program. Its value was clearly established in the Army hearing centers. It could be equally valuable in civilian practice, though unfortunately it is seldom carried out. This is curious, since, as Silverman [35] pointed out, its usefulness was first recognized in 1761, when Ernaud demonstrated an increase in auditory appreciation in a selected class of deaf children after a period of training and claimed that such training could be made of general value.

34. Macfarlan, D.: Testing Speech-Hearing and the Efficiency of Hearing Aids, Ann. Otol., Rhin. & Laryng. **57**:444-452 (June) 1948.

35. Silverman, S. R.: Training for Optimum Use of Hearing Aids, Laryngoscope **54**:29-36 (Jan.) 1944.

Silverman's own demonstration was carried out on 7 persons between the ages of 24 and 65 years who had worn hearing aids from two to thirty years. All had had a natural understanding of speech prior to their hearing loss and all had had the maximum routine adjustment and accommodation of their instruments. After they had been tested individually for perception of words and sentences, they were trained weekly for ten weeks, for an hour at a time, in the proper use of their aids. The windows of the classroom were purposely left open, so that natural surroundings would be recreated and so that training could include attention and the disregard of masking sounds. Speech was presented from electric transcriptions, to eliminate lip reading. A variety of untrained male and female voices were used for words, unrelated sentences and connected passages.

At the end of the instruction period, 6 of the 7 students had improved in word comprehension from 0 to 36 per cent and in sentence comprehension from 8 to 52 per cent. Most gains were appreciable. The single person who showed no improvement originally had no appreciation of speech through a hearing aid. The fact that these results were secured by group training is not the least important part of Silverman's demonstration in that it shows how practical such training can be.

Lip Reading.—One of the important activities of an aural rehabilitation clinic is the coordination of the training of the deafened patient. Combined training in the use of the hearing aid and in lip reading is a particularly practical phase of the program. Berry [80] cited Hudgins' experience at the Clarke School for the Deaf to prove this point: Twenty-six children, with an average hearing loss of 78 decibels, were used in the study. Their score for the use of hearing aids alone was 23 per cent and for lip reading alone (in which they had had their major training) was 43 per cent. When both the hearing aid and lip reading were used in the test, the score was 65 per cent.

The results secured with these school children are no surprise to those of us who participated in the Army (and Navy) aural rehabilitation program. Within a period of a few weeks the deafened casualty was supplied with a hearing aid, trained in its use, and taught lip reading as well as residual hearing. Formal instruction was given in the classroom and situations were contrived outside of it in which the man used his aid and practiced his new art of lip reading in the actual circumstances in which he would later be placed in real life. There are, unfortunately, no complete statistics as to how these men have succeeded in their later adjustment, but every otologist who participated in the program can recall case after case in his individual experience in which the results of this combined training were almost incredibly good.

Direct Speech–Hearing Testing.—Pure tone testing is naturally part of the scientific study of the deafened person, but the logical method of

testing a patient's ability to hear words is to test him with words, and the rediscovery of this method was one of the practical advances made in the management of deafness during World War II. Speech audiometers have been generally available for several years, and it is perfectly possible, as Macfarlan [34] pointed out, to conduct an accurate and useful test by playing to the patient records of varying intensities of sound made up of carefully selected words and numbers, which the patient repeats to the examiner. The test permits no such personal errors as are possible and probable with tests of pure tone. The word is either repeated correctly or is repeated incorrectly, and the examiner is left with no doubt about the results, except for the difficulties in calibrating speech and the proper selection of test material.

Testing hearing aids with the unmonitored spoken voice is the least reliable method of determining their relative values for the special patient. On the other hand, testing with controlled word tests (selected words delivered with varying intensities) supplemented, if desired, by sentences, is an entirely adequate method. It should be borne in mind, in weighing the respective values of word testing and sentence testing, that the use of words eliminates as much as possible the factors of education and intelligence, while the use of sentences, even if they are carefully graded, introduces these factors. It is well to carry out all tests of this kind in a soundproofed room, but as a practical matter, measured intensities of mixed noise should also be used for the obvious reason that daily living requires that speech be heard against a background of noise.

Macfarlan [34] has called attention to the value of direct speech–hearing testing in the determination of attention deafness. The test is begun with the phonograph turned up high, to secure the patient's undivided attention. After a dozen or more words have been played, it is lowered as much as 15 decibels. If the cause of the patient's auditory impairment is demonstrated by this test to be in part lack of attention, the otologist can be certain that by attention training he can improve the hearing by as many decibels as the test has indicated.

Psychologic and Psychiatric Care.—The patient with nerve deafness, like any other deafened person, may need both psychologic and psychiatric investigation. The Army experience promptly proved that this type of investigation was an important phase of the aural rehabilitation program, and the same is true in civilian life. In fact, an analysis of every patient, to determine the possible existence of emotional, attitudinal and other factors which might militate against rehabilitation would be extremely useful. It is important, however, that this phase of the program should not be overemphasized. A great deal of harm could be done by making into psychiatric problems patients who are not problems at all.

The Social Adequacy Index of Hearing.—It is essential, as has several times been pointed out, that hearing loss be estimated and hearing improvement be measured by audiometric means in terms of decibels. On the other hand, the deafened person is completely uninterested in decibels and is concerned only with such methods of treatment as will enable him to hear what is said to him. His own estimate of his own improvement, however, cannot be accepted at its face value, since many subjective, and, indeed, purely emotional, factors enter into it.

I should favor the use in an aural rehabilitation setup such as I have envisaged of the social adequâcy index of hearing introduced by Walsh and Silverman,[36] which combines the patient's improvement in decibels with his improvement in terms of social adjustment. The index was devised for evaluation of the results of the fenestration operation, but if the provision of hearing aids and the whole aural rehabilitation is to have any real meaning and any practical application, the use of some such index of results as this would seem both necessary and desirable.

Walsh and Silverman define the social adequacy index as the average articulation score when phonetically balanced word lists are placed at sound pressure levels of 20, 35 and 50 decibels above the normal phonetically balanced threshold. These three intensity levels represent weak, average and loud speech and thus reproduce the circumstances of everyday life in which the patient must employ his hearing.

The threshold zone of hearing, according to these observers, extends from a social adequacy index of hearing of 67 (a hearing loss for speech of about 30 decibels, at which level deafened persons begin to have significant difficulty in their social contacts) to an index of 10 or less (a hearing loss of about 60 decibels, at which level the hard of hearing cannot understand what is said to them). The middle of the threshold zone (which represents a hearing loss for speech of about 45 decibels and a social adequacy index of about 33) is regarded as the threshold of social inadequacy. In this twilight zone gains for speech of as little as 5 decibels (that is, 10 points on the social adequacy index) are worth while.

The general use of some such standard as the social adequacy index of hearing by all groups engaged in aural rehabilitation would in itself be a distinct gain. Comparison of results is now extremely difficult. The setting up of some useful criterion such as this index would make possible the expression of results in similar terms and on the basis of similar standards and would thus achieve some of the uniformity the lack of which, as the late Dr. Walter Hughson[37] regretfully noted, was

36. Walsh, T. E., and Silverman, S. R.: Diagnosis and Evaluation of Fenestration, Laryngoscope **56**:536-555 (Sept.) 1946.

37. Hughson, W., cited by Morrissett.[29]

so serious a defect in the initial programs in effect at the three Army aural rehabilitation centers set up in the Zone of Interior in World War II. As a matter of fact, the delayed institution of uniform programs at these centers to a certain degree nullified the potential value of the management of a large number of absolutely controlled cases of deafness. No civilian program, however carefully set up, can hope to equal the potentialities of the Army program in that respect, but the establishment of some such standard as the social adequacy index of hearing would be a step in the right direction.

COORDINATION OF THE PROGRAM

Aural rehabilitation I believe to be, in essence, the most practical solution, and the only solution presently available, for handling the problem posed by the nerve-deaf private patient. For all practical purposes it is the application to his problem of methods that solved the problem of the deafened military casualty in World War II. There is every reason why the private patient suffering from nerve deafness should be similarly benefited. There is no cure for his disease and no other help for him but his prompt acceptance of the inevitable and his prompt introduction to a mode of life which involves the use of a hearing aid, the conservation of residual hearing and instruction in lip reading and in such other phases of rehabilitation as his special needs may indicate. He can never be returned to an entirely normal life, but he can live a far more normal life than most nerve-deaf patients now live if he is given the advantages of these technics.

It is ironical that the private patient, who, generally speaking, has a better background of intelligence, education and culture than most indigent patients and than many war casualties, and who could therefore make better use of aural rehabilitation, receives less consideration than any other group of deafened persons. His plight is likely to continue until otologists as a group take steps to alter it and, at the same time, to protect themselves from one more advance in the direction of socialized medicine that will necessarily come if the state must assume the care of these patients, regardless of their financial circumstances, because their private physicians will not take the responsibility.

The most important phase of the aural rehabilitation program is perhaps the coordination of all its activities. The patient is not left to his own devices to secure a hearing aid, to learn how to use it, to learn lip reading and to do whatever else he may wish to do or may not wish to do. His whole training and readjustment are the responsibility of the otologist, who is the focal point in the program. Nerve deafness is fundamentally a medical problem, and its management must remain under medical control, but the sound engineer, the psychologist, the teacher of lip reading, the teacher of speech correction and all other participants

are his co-workers, and their joint efforts multiply many times the good results which could be secured by their work as individuals. It might be added that no small part of the inspiration of the coordinated program comes from the teaching done by deafened instructors who have risen above their own disability and from the valuable help of the Leagues of the Hard of Hearing.

Although there is nothing new about the aural rehabilitation program which I have suggested, I am not aware that anyone has up to this time taken so positive a stand about its application to the problem of nerve deafness in private practice. My conviction as to its usefulness in this type of deafness is based on my experience with the futility of all methods of treatment and on my experience in the aural rehabilitation program carried out by the Army in World War II. There was nothing new about that program. The individual components had long been known. Otologists had carried out parts and lay societies still other parts. Except that the commercial element was totally eliminated, the components of the program remained much the same. The thing that was new was that it was an integrated program, in which all the personnel concerned with the patient and all the equipment necessary for his testing and training were gathered together under the same roof and were the ultimate responsibility of the otologist in charge of the program at each center. There is every reason to believe that a similar integrated program would succeed in civilian practice and every reason to believe that it would go far to mitigate the present unhappy lot of the patient with nerve deafness.

SUMMARY

1. The significant considerations of the recent literature of nerve deafness are summarized. Data are adduced to indicate the futility of all suggested methods of medical and surgical treatment.

2. Aural rehabilitation offers the only practical method of management presently available for the nerve-deaf patient, who would be benefited by a realistic facing of that fact and by a prompt introduction to the program of rehabilitation.

3. Certain practical considerations of the program, applicable to all types of adult deafness and derived chiefly from the Army aural rehabilitation program carried out in World War II, are briefly considered.

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PERIPHERAL FACIAL PARALYSIS IN FRACTURES OF THE TEMPORAL BONE

Indications for Surgical Repair of the Nerve; Report of Cases in Which the Ballance and Duel Operation Was Used

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THE TREATMENT of facial paralysis in fractures of the temporal bone has been purely conservative until recently; first, because prognosis with conservative therapy has been considered favorable and, second, because before the introduction of surgery of the facial nerve by Ballance and Duel [1] the proper treatment could not be instituted.

Recent investigations, however, have shown that even if prognosis is good in most cases, there is, nevertheless, a limited number of cases in which the nerve ought to be repaired, and reports are available indicating that this has been done with success. The aim of this paper is to outline the indications for surgical intervention on the facial nerve.

According to most authors, a fracture of the temporal bone is present in practically all cases of closed head injuries which are accompanied with facial paralysis even if the fracture may be difficult or impossible to demonstrate in roentgenographic studies; and speaking of facial paralysis in closed head injuries will be synonymous with speaking of facial paralysis in fractures of the temporal bone.

FRACTURES OF THE TEMPORAL BONE

Fractures of the temporal bone involving the ear and the facial canal may be divided into two main groups, longitudinal and transverse. The longitudinal fractures are by far the commonest, for from among 144 cases, Skoog [2] found 140 longitudinal and only 4 transverse fractures, and Grove [3] found 146 longitudinal fractures and 16 transverse

1. Ballance, C., and Duel, A. B.: The Operative Treatment of Facial Palsy, Arch. Otolaryng. **15**:1 (Jan.) 1932.

2. Skoog, T.: Late Meningitis in Fracture of Labyrinth, Acta oto-laryng., 1949, supp. 75, p. 138.

3. Grove, W. E.: Skull Fractures Involving the Ear: A Clinical Study of Two Hundred and Eleven Cases, Laryngoscope **49**: (*a*) 678 and (*b*) 833, 1939.

fractures of the petrous pyramid. In his excellent and exhaustive paper, Grove described these fractures from different points of view:

> The course of the longitudinal fracture is quite typical. This fracture, parallel to the long axis of the pyramid is usually due to a force applied to the temporoparietal region; descending from this region, the fracture passes in front of or at the level of the external auditory canal and ends at the anterior lacerated foramen or the region of the Gasserion ganglion. . . . It courses along the anterior, aspect of the pyramid to the fossa of the Gasserion ganglion, passing through the superior wall of the Eustachian tube and the carotid canal. The middle ear is always damaged. The drum is torn, and bleeding occurs. . . . The incus may be dislocated and the ossicular ligaments torn. The labyrinth capsule remains undamaged, although marked perilymphatic hemorrhage in the scala tympani may take place, particularly near the round window.[3a] [The hearing accordingly is preserved although impaired but generally returns to normal later.]

Grove goes on to say that the facial nerve is usually spared, but the geniculate ganglion may, as pointed out by Brunner [4] and Ulrich,[5] be in the pathway of the fracture. The fracture line is in most cases large and easily discernible macroscopically, but sometimes the fissures are extremely fine and cannot be detected by roentgen rays.

Of the transverse fractures, there are two types: the internal, which transverses the internal auditory meatus, shattering the cochlea; and the external, which passes through the entire internal ear (cochlea and vestibule) and through the fallopian canal. The middle ear may be entirely spared, although a hematotympanum may occur; the drum, however, is never torn and there is no bleeding from the external canal. The fracture is accompanied with a complete and permanent loss of function, both cochlear and vestibular.

Apart from these main types, longitudinal and transverse fractures, there is one to which Ramadier and Caussé [6] drew attention: an uncommon fracture restricted to the mastoid process itself, which can open into the external auditory canal and the middle ear and also reach the facial nerve in its descending segment.

The healing of fractures of the temporal bone differs from that of other fractures, as the closure by bony callus is either incomplete or fails entirely. These fractures are repaired by fibrous tissue, especially the transverse variety, and this repair leaves indefinitely a pathway for bacteria and the danger of late meningitis.

FACIAL PARALYSIS IN FRACTURES OF THE TEMPORAL BONE

From every point of view (the immediate and delayed danger to life and the damage to cochlear and vestibular function) the longitudinal fracture has a far better prognosis than the transverse, and

4. Brunner, H.: Pathologie und Klinik der Erkrankungen des Innenohres nach stumpfen Schädeltraumen, Monatsschr. f. Ohrenh. **59**:697, 1925.

5. Ulrich, cited by Grove.[3]

6. Ramadier, J. A., and Caussé, R.: Traumatismes de l'orielle, Paris, Masson & Cie, 1937.

quite the same applies to facial paralysis which may occur in connection with a skull fracture.

These paralyses may be divided in two groups: immediate, occurring in direct conjunction with the accident, and delayed, arising after a free interval. Immediate paralyses are, as stated by Grove, usually due to an actual lesion of the nerve by the line of fracture, either in the fallopian canal or at the internal auditory meatus. They are usually complete, caused by traction, tearing, compression (blood, edema) or actual section of the nerve. According to the nature of the lesion, function may never return or the paralysis may disappear completely. The delayed paralyses appear during the first two weeks subsequent to the accident; they may be complete or incomplete, but cure results as the general rule. They probably are due to hemorrhage or edema in the facial canal or in the nerve.

According to statistics (Siebenmann,[7] Brunner,[4] Voss,[8] Ulrich [6]), longitudinal fractures caused facial paralysis in from 10 to 18 per cent of cases and transverse fractures, in about 50 per cent. Among 211 cases of skull fracture, Grove found 29 cases of facial paralysis (14 per cent); it was present in 21 cases of longitudinal fracture (18.7 per cent) and in 31.25 per cent of transverse fracture. Voss found 18 per cent of paralysis in longitudinal and 50 per cent in transverse fractures. Among 255 cases of head injuries, many without a skull fracture, Glaser and Shafer [9] found 17 cases of facial paralysis. Borden [10] found 24 cases of it among 408 patients with skull fracture, and Passow [11] reported facial paralysis in practically all fractures of the petrous pyramid.

REPORTS OF CASES OF FACIAL PARALYSIS IN FRACTURES OF THE TEMPORAL BONE IN WHICH A BALLANCE AND DUEL OPERATION WAS PERFORMED

It is generally acknowledged that the prognosis of facial paralysis in conjunction with closed head injuries is good, and because of this, but also because earlier one was not able to repair the facial nerve, treatment has been purely conservative up to recent days. By the introduction of surgical procedure for the facial nerve (Ballance and

7. Siebenmann, cited by Grove.[8]

8. Voss, O.: Die Chirurgie der Schädelbasisfrakturen auf Grund 25jähriger Erfahrungen, Leipzig, Johann Ambrosius Barth, 1936.

9. Glaser, M. A., and Shafer, F. P.: Skull and Brain Traumas: Their Sequelae; Clinical Review of Two Hundred Fifty-Five Cases, J. A. M. A. **98**:271 (Jan. 23) 1932.

10. Borden, C. R.: Otological and Rhinological Complications of Skull Fracture, J. A. M. A. **53**:429 (Aug. 7) 1909.

11. Passow, K. A.: Die Verletzungen des Gehörorganes, Wiesbaden, J. F. Bergmann, 1905.

Duel,[1] Bunnell,[12] Ney,[13] Martin[14]), new ways were paved for the treatment of facial paralysis. As Behrman[15] pointed out, there is no reason at all to put those paralyses which occur in skull fractures in a special class. As with other facial paralyses, every effort must be made to arrive at as perfect an evaluation as possible of those that occur in skull fracture and to treat them accordingly.

Even if most facial paralyses in skull fractures have a good prognosis, there is a certain number in which active intervention is needed, as illustrated by the following reports. Cawthorne[16] published a report on 4 cases, in which he mentions finding ". . . a severe fracture with fragmentation involving the inner tympanic wall and the facial nerve in its tympanic segment." By courtesy of the author,[17] I learned that in 2 cases the nerve was torn: in the first, a gap was bridged by an 8 mm. graft; in the second, the stumps, after being trimmed, were sealed together with concentrated plasma solution. In the third case, the nerve was damaged by bone pressing on it, and in the fourth, the nerve was discolored where a hemorrhage had taken place. In all 4 cases there was return of function.

Farrior and Caldwell[18] performed a decompression in a case in which, two months after the accident, there was still a complete facial paralysis and mastoiditis. The fracture of the temporal bone had extended through the roof of the antrum, and sequestrums were removed from a fistulous tract into the external auditory canal. In the infralabyrinthine area a comminuted fracture was noted immediately below the posterior semicircular canal.

> Decompression of the vertical course of the facial nerve disclosed several small spicules of bone impinging upon the medial and posterior surfaces of the facial nerve immediately below the horizontal semicircular canal. These spicules were removed. The underlying portion of the facial nerve was contused and edematous.[18]

12. Bunnell, S.: Surgical Repair of the Facial Nerve, Arch. Otolaryng. **25**:235 (March) 1937.

13. Ney, K. W.: Facial Paralysis and the Surgical Repair of the Facial Nerve, Laryngoscope **32**:327, 1922.

14. Martin, R. C.: Intratemporal Suture of the Facial Nerve, Arch. Otolaryng. **13**:259 (Feb.) 1931; Surgical Repair of the Facial Nerve, ibid. **23**:458 (April) 1936; Recent Experiences with Operation on Facial Nerve, ibid. **32**:1071 (Dec.) 1940.

15. Behrman, W.: Surgical Treatment of Peripheral Facial Paralysis in Fractures of the Cranial Base, Acta oto-laryng. **37**:189, 1949.

16. Cawthorne, T.: Peripheral Facial Paralysis: Some Aspects of Its Pathology, Laryngoscope **56**:653, 1946.

17. Cawthorne, T.: Private communication to the author.

18. Farrior, J. B., and Caldwell, P. C.: Facial Nerve Paralysis Resulting from Fracture of the Temporal Bone: Report of a Case, New Orleans M. & S. J. **100**:23, 1947.

Behrman reported a case of transverse fracture with complete facial paralysis and destruction of the labyrinth. Two months after the paralysis he explored the nerve and observed a fracture through the ampulla of the lateral semicircular canal and the facial canal. The nerve was dilacerated and the continuity broken. A graft was inserted, and facial movement was regained. To these reports the following case is added:

On Feb. 29, 1948, a man aged 34 was brought to a hospital after an automobile accident; he was unconscious, with hemorrhage from both ears and the mouth, bilateral facial paralysis and paresis of the abducens nerves but with no external lesions. The following day he regained consciousness, and on examination his hearing was found to be impaired on both sides; he said that his sense of taste was normal, but it was not tested. Roentgenograms of the right side showed multiple fracture lines at the base of the petrous part of the temporal bone, probably involving that bone. At otologic examination on March 16, clots and dried secretions were found in the external canal of both ears. Whisper was heard at a distance of 20 cm. in the right ear and at 40 cm. in the left. There was no spontaneous nystagmus. The ears were not cleaned, but six days later pus was seen in the right external canal, the drum membrane was red and swollen, with a central perforation, and the soft tissues behind and above the ear were slightly infiltrated and tender. Whisper was heard at a distance of 5 cm. in the right ear. Treatment was started with 100,000 units of penicillin administered three time a day, and the otitis soon subsided. On April 28, two months after the accident, the electrical tests showed a partial reaction of degeneration (faradic reaction negative, galvanic reaction positive, quantitatively normal but qualitatively altered). The neurologist concluded that the continuity of the nerves was not entirely interrupted and that the paralyses were probably due to a hemorrhage in the facial canals. As there were no signs of return of function, he advised a decompression in order to accelerate recovery. The patient accordingly was transferred to Frederiksborg County Hospital.

On his arrival, it was noted that he had complete bilateral peripheral facial paralysis and paralysis of the abducens nerves. Bell's symptom was present, and the right ear was dry, but the part of the drum adjacent to the malleus was swollen and red and bulged outward. There were no perforations. The left ear was normal. Whisper was heard at a distance of 30 cm. in the right ear and at 4 meters in the left. There was no spontaneous nystagmus and no sense of taste on the anterior two thirds of the tongue on either side. The faradic reaction was negative and the galvanic was positive but was qualitatively altered on both sides. The rest of the examination revealed nothing abnormal.

The indications for operation on the right side were obvious and were as follows: complete peripheral facial paralysis of two months' duration in direct connection with a fracture of the skull and roentgenograms showing various fracture lines at the base of the petrous pyramid, probably involving the temporal bone. The highly abnormal drum membrane, impaired hearing, loss of taste and hyperlacrimation suggested a lesion in or near the middle ear distal to the geniculate ganglion; since the reaction of degeneration was (partly) positive, the lesion was suspected to be severe. On the left, hyperlacrimation and the loss of taste

suggested a lesion distal to the geniculate ganglion on this side also. There was no improvement on this side either, and as the reaction of degeneration also was (partly) positive, this lesion too was suspected of being severe, and a decompression was justified, even if the possibility of a spontaneous recovery could not quite be excluded.

On May 5, nine weeks after the accident, a decompression after the method of Ballance and Duel was performed on the right side. A fracture was noted through the tegmen tympani and the medial wall of the aditus ad antrum, involving the

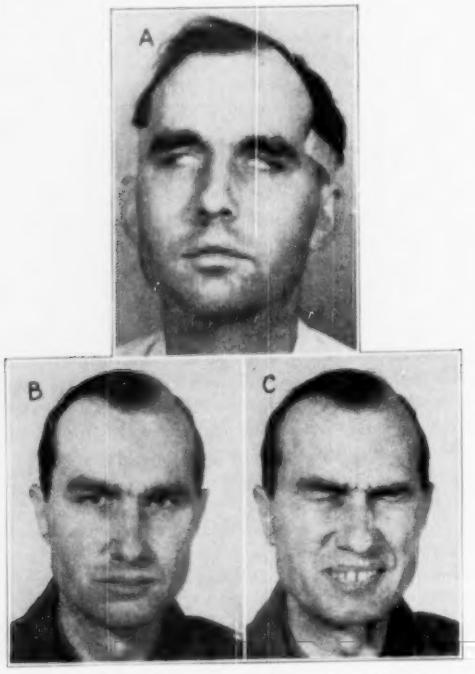

Fig. 1.—*A*, fracture of the base of the skull with a bilateral complete facial paralysis. Two months later a decompression of the nerves was performed. On the right side the facial canal was involved in a fracture and the nerve heavily compressed; on the left nothing abnormal was noted macroscopically. Rapid recovery occurred. *B* and *C* were taken seven months after the operation.

facial canal at the bend, where the tympanic part of the nerve joins the vertical segment. The fragments were dislocated and the nerve heavily compressed but not torn. The incus was displaced downward and forward into the tympanic cavity, pushing the malleus outward (and responsible for the otoscopic findings). As a result of the previous otitis media, the attic was filled with granulations. A radical mastoidectomy was done and the nerve uncovered from the geniculate

ganglion to the stylomastoid foramen. In the tympanic segment, between the bend and the geniculate ganglion, a hemorrhage had taken place in the nerve, whereas the vertical section was macroscopically normal.

After the nerve sheath was split, the tympanic part of the nerve bulged heavily.

A week later a decompression was done on the left side from the stylomastoid foramen to the bend at the lateral semicircular canal, but nothing abnormal was found, either in the osseous parts or in the nerve.

Recovery was uneventful. The day after the operation on the left side, the first signs of mobility were noted at the left corner of the mouth, and improvement was rapid. On the right side an increase of tonus and a paresthesia were noted five days later, and the first signs of mobility were noted eight weeks after the operation. On reexamination seven months later, the paralyses could not be noted when the face was at rest; the patient could wrinkle his forehead, screw his eyes up and smile and laugh, but his mimicry was somewhat stiff. He was, however, still improving. The paralysis of the abducens nerves had disappeared. Whisper was heard at a distance of 10 cm. in the right ear and at 6 meters in the left and conversation at 3 meters in the right ear and 6 meters in the left. Apart from the injury to the facial nerves, there were no disorders of any kind resulting from the head injury.

Epicrisis.—A man aged 34 was admitted to a hospital in an unconscious state, with bilateral peripheral facial paralysis, bilateral paralysis of the abducens nerves and bleeding from both ears and the mouth, as the result of an automobile accident. His hearing was somewhat impaired, but there were no vestibular abnormalities. Three weeks later suppurative otitis media developed on the right side but quickly subsided during penicillin treatment. Roentgenograms showed multiple fracture lines near the base of the right petrous part of the temporal bone, which was probably involved. Two months later there were still no signs of improvement, and since the reaction of degeneration was partly positive, a massive lesion, probably a hemorrhage in the facial canals, was suspected. On the right side the possibility of a dilaceration of the nerve could not be excluded. Accordingly, a decompression was advised to accelerate recovery nine weeks after the accident. On the right side there was a fracture involving the facial canal near the bend of the nerve at the posterior part at the lateral semicircular canal, and the fragments were dislocated, compressing the nerve heavily. The incus was displaced into the tympanic cavity, pressing the malleus outward. The nerve was uncovered from the geniculate ganglion to the stylomastoid foramen, and a hemorrhage was discovered in the tympanic segment. On the left side nothing abnormal was found macroscopically. Recovery was uneventful, and the first signs of mobility were on the left side and were noted the day after the operation; mobility on the right side was noted eight weeks later, and on reexamination seven months later the mobility of the face on both sides was good. Mimicry, however, was somewhat stiff, but the patient was still improving. The paralysis of the abducens nerves had completely disappeared.

METHODS FOR SELECTING CASES OF FACIAL PARALYSIS IN FRACTURE OF THE TEMPORAL BONE FOR A BALLANCE AND DUEL OPERATION

The cases of Cawthorne, Farrior and Caldwell, Behrman and that of the present report have shown that operation on the facial nerve is definitely indicated in some cases of facial paralysis in closed head injuries, even if the number is limited. The difficulty is to select the proper cases, and in order to accomplish this task it is of value (*a*) to analyze the prognosis of facial paralyses occurring in closed head injuries conservatively treated, and (*b*) to determine whether the results of experience with facial nerve surgery in another field, such as post-operative paralyses of otic origin, can be of some help.

Only few reports are available dealing with the prognosis of facial paralysis in closed head injuries, among which Turner's [19] paper must be mentioned first. He made a reexamination in 70 consecutive cases; in 36, the paralysis had occurred immediately and in 34 it had been delayed in onset. In 27 of the 36 cases with immediate paralysis, the paralysis disappeared completely; in 6, however, it disappeared only partially, and in 3, after eighteen to twenty-four months of observation, the paralysis was still complete. This means that in 9 out of 36 cases, or 25 per cent, recovery was only partial or did not occur at all. In 32 of the 34 cases of delayed paralysis, however, there was complete recovery, and in 2 recovery was partial. Turner suspected ". . . that a traumatic facial palsy is always associated with a fracture in the petrous part of the temporal bone, though this is not always demonstrable in X-ray studies, especially when the fracture line is in the longitudinal axis of the bone." [19] Unfortunately, in Turner's short report, many details on the nature of the separate cases are lacking.

Grove published reports on 21 cases of facial paralyses in longitudinal fractures and 5 on the transverse variety. The paralysis was complete and permanent in 1 case of transverse fracture and in 5 cases of longitudinal fracture; in the remaining cases, it either improved or cleared up completely. Of these cases, 16 were those in which the paralysis had been immediate; the rest were those in which the paralysis had been delayed in onset. The diagnosis of fracture of the petrous bone was made in all but 2 of the cases in which facial paralysis developed, and in these 2 such a type of fracture could not be excluded.

Lang [20] reported on 222 cases of fracture of the base of the skull; of these, 83 were accompanied with facial paralysis, 75 per cent of which cleared up, but in 25 per cent *zurückgebliebene Schädigungen* were encountered.

19. Turner, J. W. A.: Facial Palsy in Closed Head Injuries: Prognosis, Lancet **1**:756, 1944.

20. Lang, H.: Beobachtungsergebnisse innerhalb von 8 Jahren bei 2019 Schädeltraumen mit 222 Schädelgrundbrüchen, Beitr. z. klin. Chir. **172**:101, 1941.

It is clear from the statistics and from the case reports mentioned here that the prognosis of facial paralysis in closed head injuries is not so good as it is generally accepted to be and that this applies especially to the immediate paralyses. The greatest doubt about a favorable prognosis arises in cases of paralysis that occur with transverse fracture, because the fracture may have involved the facial canal and damaged the nerve heavily. The prognosis in cases of delayed paralysis is much better, because an interruption of the continuity of the nerve can be excluded; but even among these there were cases in which recovery was incomplete or did not occur at all.

The other method of selecting for operation proper cases of facial paralysis in closed head injuries is to make a comparison with postoperative otogenous paralyses. Such a comparison showed that in otogenous paralyses too the prognosis of the delayed paralyses generally was favorable, but that the immediate paralyses was very grave. Kettel [21] analyzed the condition in 264 patients conservatively treated; in 111, the paralysis appeared in direct conjunction with a simple or radical mastoidectomy, and in approximately 60 per cent of the patients, the paralysis remained complete on reexamination years later. In selecting those cases in which a destruction of the continuity of the nerve definitely could be ascertained, it was found that 100 per cent of the patients still had complete paralysis, and this was later confirmed by Riskær's [22] investigations.

On the other hand, it is known from numerous reports (among which those of Ballance and Duel,[1] Cawthorne,[23] Tickle,[24] Sullivan,[25] Lathrop,[26] Farrior [27] and Kettel [28] should especially be mentioned) that if the facial nerve is repaired at the site of injury the issue is very favorable. In 50 consecutive cases of nerve graftings and nerve sutures,

21. Kettel, K.: Facial Palsy of Ototic Origin, Arch. Otolaryng. **37**:303 (March) 1943.

22. Riskær, N.: The Course of Otitic Facial Palsy Under Adequate Treatment of the Disease of the Ear, Acta oto-laryng. **34**:280, 1946.

23. (*a*) Cawthorne, T.: Nerve Grafting in Facial Paralysis, Tr. M. Soc. London **60**:171, 1938; (*b*) Traumatic Facial Paralysis, n. Press **207**:260, 1942; footnote 16.

24. Tickle, T. G.: Surgery of the Facial Nerve in Three Hundred Operated Cases, Laryngoscope **55**:191, 1945; The Surgical Treatment of Facial Paralysis, in Nelson Loose-Leaf Surgery of the Ear, New York, Thos. Nelson & Sons, 1938, p. 365.

25. Sullivan, J.: Ear, in Modern Practice in Diseases of the Ear, Nose and Throat, vol. 2, sect. 1, chap. 8; to be published.

26. Lathrop, F. D.: Facial Nerve Surgery in the European Theater of Operations, Laryngoscope **56**:665, 1946; The Repair of Traumatic Lesions of the Facial Nerve Secondary to War Wounds, S. Clin. North America **26**:763 1946.

27. Farrior, J. B.: Facial Paralysis in Otology, South. M. J. **41**:348, 1948.

28. Kettel, K.: The Prognosis of Nerve Grafting and Nerve Suture in Peripheral Facial Palsies, Acta oto-laryng., 1948, supp. 74, p. 180.

Kettel [28] showed that reinnervation took place in 90 per cent of the cases. He emphasized the words of Ballance and Duel that the earlier the repair is done, the easier it is to do and the better the condition of the muscles, so the mind of the patient and the surgeon can be put at rest.

Therefore, it can be concluded from this comparison that it would be logical, and certainly desirable, to apply the same procedure that is used in paralyses of otic origin to paralyses in fractures of the skull.

DIFFICULTIES IN SELECTING CASES OF FACIAL PARALYSES IN FRACTURES OF THE TEMPORAL BONE FOR A BALLANCE AND DUEL OPERATION

The attempt to transfer treatment considered adequate for otogenous paralyses to paralyses in skull fractures meets with various difficulties: (*a*) localization and accessibility of the site of injury; (*b*) determining the nature and grade of the damage to the nerve, and (*c*) the patient's general state of health.

In postoperative otogenous paralyses the site of injury to the nerve is known approximately. In simple mastoidectomies the site of injury is almost always in the vertical segment of the nerve just below the bend at the lateral semicircular canal, and in radical mastoidectomies, at the point of the facial ridge or in the tympanic part above the promontory. All these places are situated distal to the geniculate ganglion, and, consequently, they are surgically accessible.

In skull fractures the site of injury may be located in the vertical segment of the nerve (Farrior and Caldwell), near the bend (as in the present case) or in the tympanic segment (Cawthorne, Behrman), all sites of which are accessible, but it may also be situated proximal to the geniculate ganglion in the internal auditory meatus. This place usually is regarded as inaccessible, but Tickle [24] published a report of a case (chronic otitis with sequestration of the entire labyrinth) in which the proximal end of the nerve was picked up at the level of the dura and a grafting successfully performed.

For determining the site of injury one must resort to such information as can be gained from roentgenographic studies and from noticing the extent to which the functions served by the facial nerve (sense of taste, salivation, lacrimation) are interrupted, as outlined by Cawthorne [23b] and, especially, by Tschiassny.[29] As Cawthorne [16] points out,

> Skull fractures in which facial paralysis is accompanied by signs of damage to the ear, particularly if the middle ear is affected, should be considered as possible instances of fracture involving the middle ear cleft and the facial nerve as it runs along its inner wall.

29. Tschiassny, K.: The Site of the Facial Nerve Lesions in Cases of Ramsay Hunt's Syndrome, Ann. Otol., Rhin. & Laryng. **55**:152, 1946.

One of the most difficult tasks is to determine the nature and grade of the lesion of the nerve. From the degree of the otogenous paralyses, whether partial or complete, nothing could be deducted concerning the prognosis (Kettel [21]). Many authors rely on the faradic reaction, considering a negative response a sign of a severe lesion of the nerve (Ballance and Duel,[1] Tickle,[34] Cawthorne,[28] Collier,[30] Lathrop,[26] Sullivan [25]), whereas others (Rasmussen,[31] Violé,[32] Kettel [33]) have great doubt about its value. I have repeatedly stated that a positive response may be a good prognostic sign but that nothing final can be deducted from a negative reaction. A negative faradic reaction combined with a qualitatively altered galvanic reaction (reaction of degeneration) is of more value, but undoubtedly electromyography (Weddell, Feinstein and Pattle [34]) will be the investigation of choice in the future. It may be learned that a complete block has taken place, but nothing may be learned about the nature of the lesion (dilaceration of the nerve or a severe hemorrhage), the prognosis of which may differ widely; or it may be learned that part of the axons have been interrupted but not whether they will recover function. Feinstein [35] made an examination in 12 cases of facial paralysis in closed head injuries; in 4, there was no evidence of denervation, and in each complete recovery was made; but in each of the other cases, a varying number of axons had been interrupted. Recovery occurred, but associated movements were present in every instance. Electromyography indicates the grade of the lesion but reveals nothing about the nature, and because of this its value is limited.

TREATMENT OF FACIAL PARALYSES IN FRACTURES OF THE TEMPORAL BONE AND INDICATIONS FOR A BALLANCE AND DUEL OPERATION

In otogenous postoperative paralyses, however, the choice of treatment is not difficult, since 60 per cent of all immediate paralyses conservatively treated remained complete. According to most authors with experience in this field of surgery, immediate exploration in all such

30. Collier, J. Y.: Discussion on the Limitations of Operative Treatment in Traumatic Facial Paralysis, Proc. Roy. Soc. Med. **34**:575, 1941.

31. Rasmussen, H.: Otogenous Facial Paralysis: Especial Consideration of Therapy, Nord. med. (Hospitalstid.) **28**:2214, 1945.

32. Violé, P.: Experiences in Surgery of the Facial Nerve, Laryngoscope **54**:455, 1944.

33. Kettel, K.: Bell's Palsy, Arch. Otolaryng. **46**:427 (Oct.) 1947; footnotes 21 and 28.

34. Weddell, G.; Feinstein, B., and Pattle, R. E.: The Electrical Activity of Voluntary Muscle in Man Under Normal and Pathological Conditions, Brain **67**:178, 1944.

35. Feinstein, B.: The Application of Electromyography to Affections of the Facial and the Intrinsic Laryngeal Muscles, Proc. Roy. Soc. Med. **39**:817, 1946.

cases is absolutely indicated because a severe lesion of the nerve is suspected. At this time the operation is easy because the site of damage is approximately known and accessible, and should the decompression show that the nerve is not macroscopically injured, no harm has been done. It is much more difficult to select the right treatment for immediate paralyses in skull fractures, for in these instances there is a better prognosis with conservative treatment: in 75 per cent recovery is complete, whereas in 15 per cent there is partial clearance and in only 10 per cent does the paralysis remain massive. A routine exploration like that done in cases of immediate postoperative paralyses is consequently not justified, and all efforts must be exhausted to arrive at the best possible estimation of the degree of the lesion before a decompression is contemplated.

As previously mentioned, in cases of immediate postoperative otogenous paralyses the nerve ought to be repaired as soon as possible, and this can be done because the patient's general state of health permits it. In cases of paralyses occurring in skull fractures, such repair may well be out of the question, and a decompression, if indicated, must be postponed of course until the patient has recovered.

Farrior and Caldwell stated that "early exploration of any facial paralysis resulting from fracture of a temporal bone is the treatment of choice."[18] I agree that the exploration ought to be done as early as possible, but, and this is very important, provided that a diagnosis can be made of a severe lesion in a surgically accessible place. Cawthorne's statement[16] previously cited should be borne in mind. It is, however, very difficult to arrive at a final diagnosis, and in many cases one is forced to act on suspicion. In these circumstances how long shall one wait before an exploration is undertaken? From my[21] reexamination in cases of otic paralyses conservatively treated, it is known that in all cases in which the paralysis cleared up completely the first signs of recovery had been noted before two months had elapsed and that recovery was incomplete in those cases in which the initial movements had started after this period of observation. From Turner's[19] report of facial paralyses in closed head injuries, it was noted that in 24 of 36 cases of immediate paralysis the first signs of mobility occurred in from a few days to three weeks after the accident, and the paralysis in these cases cleared up completely within three to eight weeks. In 3 cases mobility started to return within eight weeks, and in these cases also the paralysis disappeared. In 6 of the remaining 9 cases the first signs of returning mobility were not noted till after eight weeks, and in these recovery was only partial; in 3 cases the paralysis remained complete. Among 34 cases of delayed paralysis, the condition in 32 cleared up completely; it was rated that the time of beginning recovery in 25 cases was within

three weeks. This means that in all cases of facial paralysis in closed head injuries, both immediate and delayed, in which the paralysis disappeared completely and spontaneously, the first sign of returning function had been noticed within eight weeks after the accident and usually much earlier, just as in cases of otogenous paralysis. For these reasons Behrman [18] suggested observing the patient for two months. If at the elapse of this time no signs of beginning recovery can be observed and the lesion is surgically accessible, an exploration of the facial nerve should be done. In this respect I quite agree with him.

Ersner [36] also recommended active intervention, as a decompression of the fallopian canal permits the escape of fluid and thus allows the edema of the nerve to subside, and repair of the nerve if the continuity is broken.

On the other hand, a conservative attitude is adopted by Turner [19] and Coleman.[37] Turner stated that his series does not provide an argument for operating unless there has been no recovery for at least six months after the injury; but I do not think he is right, for from among his 36 cases of immediate paralysis recovery was only partial in 6 and in 3 there was no recovery at all. In these 3 cases, destruction of continuity of the nerve must be strongly suspected, just as in Cawthorne's and Behrman's cases, and such a lesion ought to be repaired as soon as possible. Turner, however, has his doubts about his own statement, for a few lines later he puts the question of whether early decompression of the facial nerve in the canal in cases in which there is going to be slow recovery by regeneration could hasten recovery and prevent the development of associated movements and facial contracture.

Coleman recommended a conservative treatment of facial paralysis following fracture of the base of the skull in view of the large percentage of cases of spontaneous recovery. However, he recommended the same kind of treatment for otogenous paralysis, and in this respect I do not agree with him.

It is very important to arrive at the proper decision as early as possible, because if the repair of the facial nerve is postponed too long complete recovery is jeopardized. Atrophy of the muscles may be prevented by an adequate treatment with massage and galvanic stimulation. For this stimulation Clemmesen's myotensor is superior to any other machine, for the duration and the character of the electric impulse can be adjusted to the chronaxia of the muscles at any given moment. From the paralyzed corner of the mouth a wire splint hook held around the

36. Ersner, M. S.: Fracture of the Temporal Bone, in Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1945, p. 408.

37. Coleman, C. C.: Surgical Lesions of the Facial Nerve, Ann. Surg. **119**:641, 1944.

ear and used during the night and a cigaret holder used during the day will counteract an overstretching of the muscles. What cannot be prevented is atrophy by inactivity of the cells in the facial nucleus of the brain, which also takes place, and what this means to the final issue needs no further explanation.

If the patient has not been seen for some time, eventually years, after the injury with a partial or even complete paralysis, can anything be accomplished at that stage? If the paralysis is complete, if the site of injury can be located and if there is no atrophy of the muscles, an exploration of the nerve should certainly be undertaken. Many reports are available indicating instances in which an otogenous postoperative paralysis of long standing had been successfully repaired by nerve suture or grafting years later. There is nothing to risk and everything to gain.

Even in cases in which there is only very little function without practical significance I should also advocate an exploration. In some cases the facial nerve proximal and distal to the lesion will be connected by only a slender bundle of atrophic neurofibrils. Considering the excellent results which have been obtained in nerve grafting, it is better to resect this atrophic link and let the neurofibrils from the whole area of a fresh nerve section travel down the distal segment of the nerve through the nerve graft; this is an experience which I have gained in dealing with similar cases of postoperative otogenous paralysis. This procedure has proved superior to that of preserving the connecting atrophic strand and applying an end to side graft, a preference which is easily understood, because through a slit in the side of the nerve sheath the distal end of very few, if any, of the interrupted neurofibrils will be exposed and accordingly not have a chance to grow downward. Moreover, those fibrils which will be exposed will, as a rule, be unable to regenerate on account of pathologic alterations.

And what of the cases in which there is partial recovery to the extent that the paralyses is not noted when the face is at rest and the patient can smile a little and eventually close the eye? Can anything be expected from a decompression in such cases? It may be that by removing impinging bone spicules or by relieving pressure, the condition may improve. In similar cases of Bell's palsy the condition has improved (Flodgren,[38] Kettel [33]) or the paralysis has even disappeared completely (Bergström [39]) after the pressure has been relieved.

Following is a report of such a case:

As a result of an accident in 1946, a woman aged 39 had bleeding from the right ear and suggilations on the right mastoid process, with complete facial

38. Flodgren, E.: Rerouting of Facial nerve, Nord. med. (Hygeia) **31**:2159, 1946; Contributions to the Surgery of Facial Palsy, Acta oto-laryng., 1948, supp. 74, p. 188.

39. Bergström, E.: The Question of the Etiology and Treatment of What Is Known as Rheumatic Facial Paralysis, Acta oto-laryng. **31**:541, 1946.

paralysis. Several weeks afterward, the movements began at the corner of the mouth, but the improvement soon stopped. Nine months later she was referred to Frederiksborg County Hospital. She was able to smile a little (fig. 2 *A*), but could not close her right eye (fig. 2 *B*) or wrinkle her forehead. However, as the faradic current evoked strong contractions around the eye and of the forehead (fig. 2 *C*) it was considered justified to try to improve the spontaneous function by a decompression. In the cells between the sigmoid sinus and the posterior osseous wall, stale blood and cellular destructions were found, obviously remnants of a fracture; the facial canal, however, was macrospically normal, and the nerve did not bulge after the sheath was split. On reexamination three years later, it

Fig. 2.—*A* and *B*, slight improvement of facial paralysis before decompression. *C*, signs of contraction evoked by faradic current. *D*, condition three years after decompression.

was found that she could close the eye (fig. 2 *D*), that the movements of the mouth were as before the decompression and that the forehead was immobile. (Three similar cases in which a limited improvement was seen are available.)

In some of my cases the patient got rid of a very annoying contracture. It must be understood however—and the patient should be told in advance—that nothing definite can be promised. A complete restitution should not be expected because degenerative and irreversible alterations have taken place in the facial nerve and perhaps in the muscles

too. The decision may be very difficult to make and can only be made by a surgeon with a thorough knowledge of such cases and special training in such operations.

If the patient has recovered partially, and since the result even in the best cases of nerve grafting or nerve suture will never be restoration of facial movements in full, a resection of the damaged part of the nerve should in such cases certainly not be contemplated.

SUMMARY AND CONCLUSIONS

Fractures of the temporal bone can be divided into two main types, longitudinal and transverse, of which the longitudinal is by far the commoner and has the better prognosis with regard to life and cochlear and vestibular function. In cases of longitudinal fractures, facial paralysis was present in from 10 to 18 per cent and in cases of transverse fracture, in about 50 per cent. The paralyses may be divided into those that are immediate and those that are delayed. According to statistics, the delayed paralyses have a favorable prognosis with conservative treatment, whereas the prognosis of immediate paralysis was not so good as generally believed. Among Turner's [19] cases the paralysis cleared up completely and spontaneously in only 75 per cent.

Even if the number of cases is small, there are definite indications for surgical repair of facial paralysis occurring in fractures of the temporal bone, as illustrated by cases from the literature and by a personal report.

A survey is given of the difficulties which are encountered in selecting those cases for which repair of the facial nerve should be contemplated. I arrived at the conclusion that an exploration of the facial nerve should be done in cases of immediate paralysis as soon as the patient's general state of health permits it, provided that a final diagnosis of a severe lesion of the nerve in an accesible place can be arrived at. In many cases this is impossible, but if after two months of observation there is no sign of returning function a severe lesion must be suspected, and, accordingly, a decompression is indicated.

In the majority of cases of delayed paralysis, the first signs of returning mobility will be present within two months; should this not be the case, treatment similar to that of immediate paralysis must be instituted.

In cases of long-standing paralyses with very little or no facial function an exploration should be done, provided the muscles have not atrophied and the site of damage can be located; there is nothing to lose and everything to gain. If, however, the patient has recovered partially and the function is fairly good, the indications for decompression should be narrowed considerably; a limited improvement may take place, but it is impossible to predict the outcome.

If the place of injury is surgically inaccessible an anastomosis between the facial and another cranial nerve should be carried out. It is stressed that operations on the facial nerve should be undertaken only by otologists who have been trained for this special work.

In all cases, in which either conservative or surgical treatment is used, the function of the muscles must be kept alive by means of massage and galvanic stimulation, for which Clemmesen's myotensor is the machine of choice, till reinnervation has taken place. A wire splint hooking from the paralyzed corner of the mouth, a wire splint hooking held around the ear and used during the night and a cigaret holder used during the day will counteract an overstretching of the muscles.

ADDENDUM

Torre [40] has operated in 2 cases of facial paralysis with fracture of the petrous pyramid. In the first case the descending part of the nerve looked very abnormal; in the second case the nerve was compressed by fragments near the bend at the posterior end of the lateral semicircular canal.

Slotsgade 34.

40. Torre, F.: Apropos du traitement chirurgical des paralysis faciales secondaires aux fractures du Rocher et de la mastoide. Afrique franc. chir., May-June, 1947, pp. 182-186.

PREVENTION OF DEAFNESS

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THE PREVENTION of deafness should be a constant and major objective in otology, both before any sign of trouble is evident and after poor hearing has supervened. At all levels of hearing loss, prevention is of primary importance.

At first thought it would appear that there is no problem when there is no deafness. How can one prevent something which shows no sign of appearing and, by common experience, does not therefore impend? Moreover, first signs are faint, so faint that they flash no clear warning of disaster. They may arise from many widely separated parts of the body and from many different causes, any one of which may not and generally does not cause any deafness whatsoever. Nevertheless, the sure way is to take preventive measures early and, when possible, before any signs of deafness appear. This is the challenge, and slowly but surely medical knowledge and skill have progressed to a point where this can be done; in fact, the tools now available for prevention of deafness are not nearly so scarce as are the efforts to use them and to use them in time.

In this brief paper, only a superficial review of progress can be presented. Stress will be placed on the preclinical approach to the problem. From the etiologic standpoint the fundamental and activating factors underlying the different types of deafness may be grouped as follows:

Fundamental	Activating	Types of Deafness
Hereditary	Anomalies of development	Impedance External ear Middle ear
	Benign and malignant growths	Capsule (otosclerosis) Inner ear
Congenital	Traumas; rubella; poisons and drugs; diet; metabolic, endocrine; autonomic, psychosomatic and vascular disorders (anoxia)	Neural Peripheral Medullary Central (cortical)
Acquired	Infections (Bacterial and virus)	
Unknown	Allergies	Psychogenic Malingering

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All these factors are of major importance; they may interact and overlap, singly and in combination, and be affected by certain participating conditions, particularly those, preclinical and clinical, which follow: pain, tinnitus, vertigo, emotional reactions and personality and contacts (environment) of the patient. All may be present, and yet, because of strong resistance, immunity or lack of favoring environment, no deafness may occur; in which case, these conditions may remain hidden and be considered preclinical factors. Following is a discussion of some of these factors:

Heredity, as the Irishman might say, is inherited, so what can a body do about it? The fact is that inherited disorders are often controllable, because by itself heredity is not a cause. It is merely the soil in which some disorders find an environment favorable for growth, but there is no growth or activation of the lesion unless the environment, internal or external, is favorable for it. The soil of heredity is doubly favorable for deafness if both parents have the same type of deafness. It is generally unfavorable if the deafness of the parents differ. Therefore, environment should be made unfavorable for deafness to prevent so-called inherited deafness. A deaf or hard of hearing person should not marry anyone with deafness caused by lesions similar to his own. This is a real preventive measure, advisable and legitimate from the hereditary standpoint. However, do not imagine that by breeding, sterilization or any other measures, short of destruction of the whole human race, we can wholly eradicate so-called hereditary deafness. It cannot be done, because the genes in which the hereditary elements reside carry over from generation to generation, perhaps forever, whether or not they fail to manifest themselves in certain offspring or for several generations.

Congenital defects may be hereditary or acquired. Unless they are incurred through intrauterine accident, infection or disordered metabolism they are usually beyond influence, but the discovery that fetal defects may be caused by drugs and toxins in the pregnant mother opens up a new means of prevention in congenital deafness. Rubella (German measles) has been thought of as a harmless and mild disease, with rare complications and few if any sequelae. It is now known that rubella is a potent factor in congenital cataract, glaucoma, mental and cardiovascular defects, deafness of varying degrees to complete deafness and several other disfiguring and disabling defects. Rubella in the mother is especially dangerous to the fetus when the mother acquires it during the first three months of pregnancy. Almost 100 per cent of the offspring have been reported to have congenital defects if the disease is acquired during the middle of the first trimester of pregnancy. Strange to say, many other seemingly more dangerous contagious diseases appear to affect the fetus less frequently than

rubella, but of this we physicians are not certain. The young, undifferentiated tissue of the fertilized ovum appears to be extremely vulnerable to virus infection. An obvious preventive measure is to protect the mother from exposure to infection of all kinds, especially during the early months of pregnancy.

The danger of acquired factors need not be stressed to the informed physician. They are constantly in the offing and are understood far better than congenital factors; they strike often during childhood. Dramatic and exciting is our ability now to diminish the severity and even to prevent most of the diseases of childhood.

Rubella in the infant appears to be much less dangerous than rubella in the fetus, and it often may be wise to expose healthy female children to such a mild disease as rubella so that they may acquire immunity and thereby in adulthood not endanger the preborn child.

Scarlet fever, even mild scarlet fever, may develop dangerous sequelae. The Dick test and the newer drugs have revolutionized treatment, and now, by specific treatment with antitoxins or globulins, we can, if we act in time, prevent the sequelae prone to cause aural complications and deafness.

Pertussis (whooping cough) is an extremely serious disease, especially in infancy, because of its high mortality rate and crippling complications. The mother, even though she may have had pertussis, possesses little ability to transmit immunity to the fetus. Hemophilus pertussis or the parapertussis organism is the etiologic factor. Its presence in the sputum definitely determines the diagnosis. Also of value for the diagnosis is pronounced lymphocytosis, at times accompanied with hyperleukocytosis. The skin tests are invaluable for indicating susceptibility or weak immunity. Active immunization may be satisfactorily obtained by prophylactic injections of whole antigen, which is well tolerated even by the infant of 2 or 3 months. Immunization, however, is not maintained for long periods. Treatment with hyperimmune serum and globulin has brought the mortality rate down to about 1 per cent, and the incidence and severity of complications have been definitely diminished by sulfonamides and penicillin.

Mumps, long a mystery, is now known to be due to a virus and can be diagnosed by complement fixation tests. The skin tests are also valuable, even in the absence of glandular swelling. Mumps is a serious disease. It frequently causes total deafness by destroying the neural mechanism of the ear. A common complication of mumps is meningoencephalitis, and this may be differentiated from the other types of brain fever, all of which, however, frequently cause nerve deafness, even in the absence of any definite diagnostic signs. Treatment by convalescent serum is helpful if administered in time. Experimentally,

certain polysaccharides seem to have preventive powers. Most important is to put the child to bed and to keep him there until all danger of complications has passed.

In measles, brilliant advances have been made in the use of convalescent serum as a therapeutic and prophylactic agent. The globulin fraction, especially the gamma globulins, which carry the immunizing agents, constitutes one twenty-fifth of whole serum and rarely produces unpleasant reactions. Permanent immunity may be built up by administering less than a surely preventive dose so that the child may be allowed to have a very mild illness. Larger doses will prevent the disease entirely when this is desirable. The dose for modified immunity is about one fifth that for absolute prevention. The sulfonamides and penicillin are of inestimable value in the treatment of complications, especially of the complications involving the ear. See to it that you, the otologist, stress the preventive treatment to the physician in charge. But if the response is poor do not delay operating. A timely operation is the primary preventive treatment, so far as the preservation of hearing is concerned.

Encephalitis and meningitis as a complication of measles, or of any infection, has in the past been highly fatal, and when not fatal, a major cause of extreme or total deafness. Now, if at the first indication of labyrinthine or brain involvement massive doses of penicillin (500,000 units have been successfully used) and sulfonamides are intensively administered it is possible to control the inflammation before irreversible deafness supervenes. Treatment should be started immediately; if in doubt, do not wait for the laboratory report. Even epidemics of meningococcic meningitis have been brought under control by the prophylactic use of sulfonamides.

In the treatment of lesions causing deafness there is the danger of relying too much on methods which, although scientifically accurate, fail to recognize and utilize the advances available in other fields of medicine. For instance, the effects of irradiation may be enhanced by the use of antibiotics, and antianemic drugs, hormones and vitamins are valuable aids to irradiation and surgical treatment. Keep in mind that in some instances our so-called wonder drugs have led us into false security and that they may provoke violent reactions and cause deafness instead of preventing it. If you really wish to prevent deafness caused by otitis media do not treat it routinely or over the telephone.

That there are many unknown factors in deafness is evident. There will always be unknown factors in every ill to which the flesh is heir, but they are few now as compared with former times. I shall mention here the great unknown in otology, otosclerosis, and discuss possible means for its prevention. The use of surgical treatment to overcome

or circumvent some of the hearing loss is widely known, but, statements to the contrary notwithstanding, so far as is known, it in no measure cures the disease or prevents its progress. It has no effect whatsoever on the sclerotic bone of the lesion or on the ankylosis of the stapes, the cause of the conduction deafness. It has not been suggested for prevention of the disease (as yet).

We know a great deal about the appearance of the bone in otosclerosis; we know how it looks under high power magnification and by polarized and ultraviolet light. From its tiniest beginnings to its largest development, this picture has been often described, but its microscopic morphologic structure does not reveal its cause. We know much about its inheritance and how it frequently appears to skip a generation or so, but intensive research has failed to produce any widely accepted hypothesis for its cause.

Clinically, certain observations and laboratory findings seem to be correlated to the activation of the lesions and the increases in deafness. Among these, emotional and endocrinal disturbances and severe illnesses are outstanding. Experimentally, a multitude of bone dyscrasias may be caused by hormonal imbalance and food deficiencies, but all efforts to produce typical otosclerosis in animals by these or any other means have failed. There must be a basic biologic reason for such failures; they are, I believe, due in part to the more rugged or stable emotional status of the lower animals, their lower level of cerebration and their less prolonged emotional reactions. Also, these failures are primarily due to the absence of the inheritance factors in animals; their ancestors never having had otosclerosis, they cannot have inherited any predisposition to it. This also holds for human beings who have not inherited a definite predisposition to it. They too may be subjected to the identical factors which in others are associated with activating factors, and yet they do not acquire otosclerosis. Until we are able to produce it in animals, I shall hold to the opinion that otosclerosis cannot be produced in human beings unless there is an inheritance favorable to it. It is our continuing task to find means for determining the activating factors involved.

Our ignorance and our many failures should not deter us from attempting to find methods of prevention. Families prone to otosclerosis should be informed of present knowledge, meager though it be. Preclinical prognosis and preventive treatment are our major aims, because at this time no reversal of the process seems attainable.

This is what I do. Even if there is no suspicion of deafness, in every instance I make a thorough and complete examination, including family and personal medical histories; remember that you are looking for leads and faint signs. In my opinion it is imperative that every

child in otosclerotic families receives a thorough otologic and comprehensive medical examination with laboratory check-up. For adults and older children I include determinations of the basal metabolic rate, blood cell and differential count, estimations of the serum calcium, phosphorus, cholesterol and chlorides, plus additional studies when indicated.

Signs and symptoms, which in other types of deafness may be of little or no import, appear significant when preclinical or early diagnosis of otosclerosis is the objective: 1. One sign is a loss of even 10 to 15 decibels indirectly to the lower frequencies without apparent cause —even a lesser loss if it is slowly increasing. In children and adolescents this means a loss of only 5 or 10 decibels below the average normal at these frequencies, as shown on audiometric charts, because the normal threshold line on the chart is 10 to 15 decibels below the average normal for children who have no loss of hearing. The testing is done in a well soundproofed room. 2. Another sign is tinnitus, even if it is so faint or inconstant that the patient is hardly aware of it. 3. There are certain abnormalities detectable by otoscopy which sometimes precede deafness from otosclerosis—a pinkish glow on the promontory, the presence of exostoses and the absence of wax and hairs in the external auditory meatus. 4. Abnormal vascular and cough reflexes are other signs. 5. The color of the sclera should be noted. 6. Emotional shocks or emotional instability frequently precedes the onset and the progressive phases of deafness from otosclerosis. Inquire carefully into these matters and about the age at first menses; the diet habits during infancy, childhood and adolescence; the place of birth, and the type of water and milk received. Knowledge concerning the mother's diet and health during pregnancy and lactation is also important. Anything that might affect the metabolism of the osseous and neural tissues of the body should be carefully explored. The endocrinal function and the mineral metabolism should be investigated and stabilized.

No one sign alone is sufficient for a preclinical or postclinical diagnosis, but when several are present they become important, especially so in otosclerotic families. For instance, at any examination of more than 600 of my patients, blue scleras were present in more than two thirds of all those with otosclerotic deafness; whereas in the controls with normal hearing only about 10 per cent showed blue scleras. This does not mean that the otosclerosis or its severity was necessarily closely related to the blueness of the sclera, but it does show that there may be some relation between the two conditions.

The reliability of some of these signs and symptoms as diagnostic criteria is uncertain; it varies from 30 to 70 per cent. This percentage is not too bad when compared with the reliability of the tests used in

many other diseases. Even with these imperfect criteria, we can by trial and error and constant endeavor evolve preclinical diagnostic technics and thereby advance our means for the prevention of deafness. The only sure way to avoid accidents is to prevent them. Fundamentally, disease is an accident and deafness is an accident. The only sure way to avoid deafness is to prevent it. The only way to avoid otosclerotic deafness is to prevent it.

The mere attempt to prevent otosclerosis or its progress may produce, and in a few instances has produced, by trial and error a greater knowledge of some of the factors seemingly correlated to the lesion and, also of importance, many of the factors seemingly having no relation to it. Such knowledge is valuable. By continuing experimentation and close observation, we have obtained several aids to diagnosis and treatment, and though they are far from being one hundred per cent reliable, I venture to state that in many instances they would be invaluable if we had a greater opportunity to examine patients before or even soon after deafness is detectable. The only practical way to accomplish this is to test routinely every child during all the school years and adults at every opportunity and to act accordingly and promptly.

It is hoped that this brief discussion has made it evident that great advances have been made in practical measures for the diagnosis and treatment of many of the diseases and disorders causing deafness and, in certain instances, advancement has been made even in preclinical diagnosis, prognosis and prevention. The major difficulty is to make physicians in general and otologists in particular more cognizant of what should and can be done for prevention by using more capably the tools now available.

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SUPERFICIAL EPIDERMOID CARCINOMA OF THE LARYNX

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THE CASE presented in this paper is of particular interest for various reasons: 1. The patient had a papillary carcinoma of the larynx, which does not frequently occur. According to the experience of McKenty, only 1 per cent of intrinsic laryngeal carcinomas have a papillary structure. 2. The carcinoma was associated with trichinosis of the laryngeal muscles, which did not produce clinical symptoms. 3. An anatomic specimen of newly formed "vocal cords" was obtained about three months after a laryngofissure was performed. 4. One biopsy specimen taken from the larynx showed a superficial epidermoid carcinoma (carcinoma in situ). All these findings, particularly the two last mentioned, warrant an exhaustive report and discussion of the case.

REPORT OF A CASE

B. B., a white man aged 62, had had in the last two years occasional common colds associated with hoarseness, which disappeared together with the cold. In January 1948 he again had a cold associated with hoarseness, but subsequent to this attack the hoarseness did not disappear. He could talk only with the whispered voice.

At admission to the hospital on Feb. 13, 1948, he spoke with an extremely hoarse voice. He had no difficulty in swallowing. He was slim and edentulous, and the nodes of his neck were not enlarged. The anterior part of the right vocal cord was thickened and yellow, and there were pointlike, hemorrhagic defects of the epithelium. The thickening of the cord extended toward the anterior commissure, but there was a distinct boundary toward the posterior part of the right cord, which was normal. During phonation, the left cord showed a slight excavation, corresponding to the thickening of the right cord. The motility was normal. Biopsy of the right cord revealed epidermoid carcinoma. On February 20 a tumor the size of a pea was noted in the anterior commissure. The tumor had an uneven surface and extended into the left vocal cord. Below the right vocal cord was a thickening covered with whitish material.

On February 21 a laryngofissure was performed according to the technic of Jackson. The tissue removed included the right true cord, part of the left cord, a great portion of the false cords on both sides and a considerable amount of subglottic mucosa on the right. Two days after the operation the patient was out of bed, and four days after the operation he was able to swallow.

From the Department of Otolaryngology, University of Illinois College of Medicine.

Fig. 1.—Oblique tangential section through the false cord (*F*) and the vocal cord (*V*); (*a*) carcinomatous part of the tumor forming a nodule; (*b*) papillomatous part of the tumor. On the left side of the picture is the anterior commissure, containing large blood vessels not invaded by the tumor.

Microscopic Examination.—The excised mucosa was stretched on a cork. After fixation of the tissue oblique tangential sections were made, and the serial sections were stained with hematoxylin and eosin. There was a diffuse tumor in the subglottic area which scarcely bulged into the subglottic space. The tumor did not invade the actual anterior commissure but extended below the commissure from the right to the left side (fig. 1). It extended upward to the edge of the true cord but did not reach it. Toward the trachea, it extended close to the margins of the specimen; in this direction, the excision apparently was not made a sufficient distance from the tumor. Backward, the tumor extended to the right vocal process. Toward the depths of the mucosa the tumor extended into the subepithelial connective tissue and to the subepithelial glands, but it reached neither the muscles of the vocal cord nor the connective tissue of the anterior commissure. The true cord was covered with stratified squamous epithelium, showing variable thickness and moderate keratinization. At several sites the basilar layer of the epithelium and the normal stratification of the epithelium were absent. However, even in these areas there was a distinct boundary between epithelium and connective tissue. Below the edge of the cord, close to the anterior commissure, the epithelium ended abruptly and was replaced with a nodule-like tumor, which extended deeply into the tissue and had a papillary structure (fig. 1, *a*). The epithelial folds were long and extended from the mesial surface lateralward as well as from the dorsal surface ventralward, forming a mass of intertwined folds. Toward the depths of the mucosa the folds frequently fused, forming a compact tumor. There was a distinct boundary between the tumor nodule and the connective tissue, the latter showing severe inflammation. The tumor nodule consisted, to a great extent, of epithelial cells. In the core of the epithelial folds there were a small amount of connective tissue and numerous capillaries, which contained a large number of leukocytes. The nuclei of the epithelial cells were variable in size; they were round or spindle-like and frequently were piled up to such a degree that there was very little space left for the cytoplasm. There were no giant cells. Mitotic figures were infrequent. There was not much keratinization, but degenerative changes of the cells were frequent. The epithelium did not show any stratification, and only occasionally several cuboid cells formed a layer which could be called a basilar layer.

For a short distance below the tumor nodule the mucosa was covered with a stratified squamous epithelium without a basilar layer. Below this area the epithelium again ended abruptly and was replaced with a tumor that did not form a nodule but showed a papillary structure (fig. 1, *b*). The papillae, which bulged slightly into the subglottic space, had cores consisting of a considerable amount of inflamed connective tissue and capillaries. The cores were covered with a squamous epithelium, which at several sites had a distinct basilar layer; at other sites the stratification of the epithelium was poorly determined. There was a moderate formation of keratin, and mitotic figures were infrequent. The epithelial crypts, which extended into the depths of the mucosa, were surrounded by lymphocytes (fig. 1, *b*). In the lumen of the crypts was keratinized epithelium or cellular debris, but there were no cornified pearls. There were only a few leukocytes in the walls and in the lumen of the crypts, because the walls offered considerable resistance to the invasion of the surrounding leukocytes.

The false cords were inflamed and edematous, but no tumor was there (fig. 1, *f*). The vocal muscle showed severe myositis, and there was disintegration of muscle fibers. There was no tumor tissue in the muscle. Throughout the muscle, particularly at its insertion on the anterior commissure, there was a deposition of

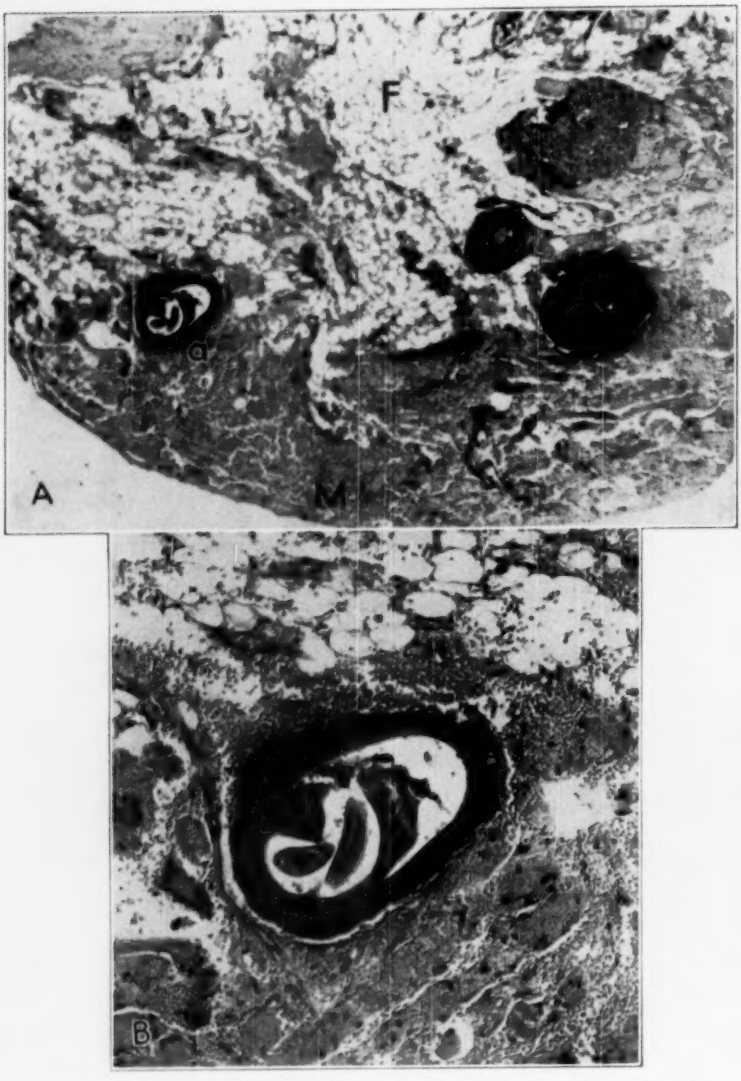

Fig. 2.—*A*, showing three trichinellas in the depths of the right vocal cord; (*M*) muscle fibers (to a great extent normal); (*F*) fat tissue; (*a*) trichinella shown in *B* in higher magnification. *B*, showing recent hemorrhage but no inflammation in the area surrounding the trichinella.

calcium between the muscle fibers, causing a necrosis of these fibers. Between the muscle fibers and in the subepithelial connective tissue there was a large number of trichinellas (fig. 2), which were piled up close to the anterior commissure and

Fig. 3.—Biopsy specimen taken on April 27, 1948. In (x) the boundaries between epithelium and inflamed connective tissue are indistinct.

close to the periosteum. The trichinellas were ensheathed by a hyaline capsule, which may or may not have been calcified. Occasionally, there was a fine connective

tissue membrane on the outside of the capsule. The encysted trichinellas were spherical at the surface of the muscle and oval in the center of the muscle, this difference in form apparently caused by the pressure of the surrounding muscle fibers (fig. 2). Regardless of whether the trichinellas were calcified or not, there was a moderate accumulation of small round cells about the capillaries, close to the trichinellas. When the trichinella was oval, the cells accumulated at the poles of the trichinella and extended far between the surrounding muscle fibers. Because of the moderate number of cells, the infiltration could be seen only on serial sections.

Clinical Course.—Subsequent to the laryngofissure, on March 4 a tumor developed in the area of the anterior commissure, consisting apparently of fibrin.

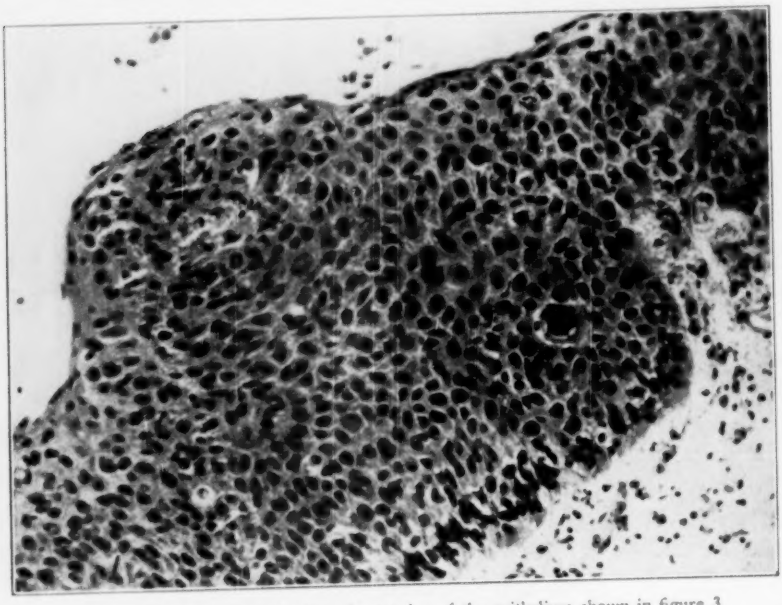

Fig. 4.—Higher magnification of a portion of the epithelium shown in figure 3, containing the carcinoma in situ.

Penicillin was administered. On March 27 both false cords were edematous and reddened and approximated on phonation. The true cords were replaced by scars. A polypoid whitish tumor was removed from the area of the anterior commissure and proved to consist of granulation tissue. On April 10 granulations were removed again from the area of the anterior commissure, and pieces were removed from the false cords. There was no lesion in the subglottic area. Microscopic examination revealed mild fibrosis and edema of the mucosa. On April 27 there was considerable thickening of the false cords in the area of the anterior commissure, but the anterior commissure was firm and there was no ulceration. Subglottically, on the right side there was a whitish mass, from which tissue was removed. Microscopically, the specimen consisted of connective tissue, which was

densely infiltrated by small round cells and by a smaller number of polymorphonuclear cells (fig. 3). The tissue was covered with squamous epithelium, which contained high papillae. Occasionally, epithelial pegs were displaced into the depths of the inflamed connective tissue. The epithelium, to a great extent, did not show typical stratification (figs. 4 and 5), but it did show numerous mitotic figures. There was moderate migration of leukocytes through the epithelium and no keratinization. The diagnosis of a superficial epidermoid carcinoma was made. On May 7 there was noted a necrotic area at the apex of both "cords," on the right side more than on the left. Both cords were hyperplastic. On phonation, the cords were imperfectly adducted and a diamond-shaped area remained patent.

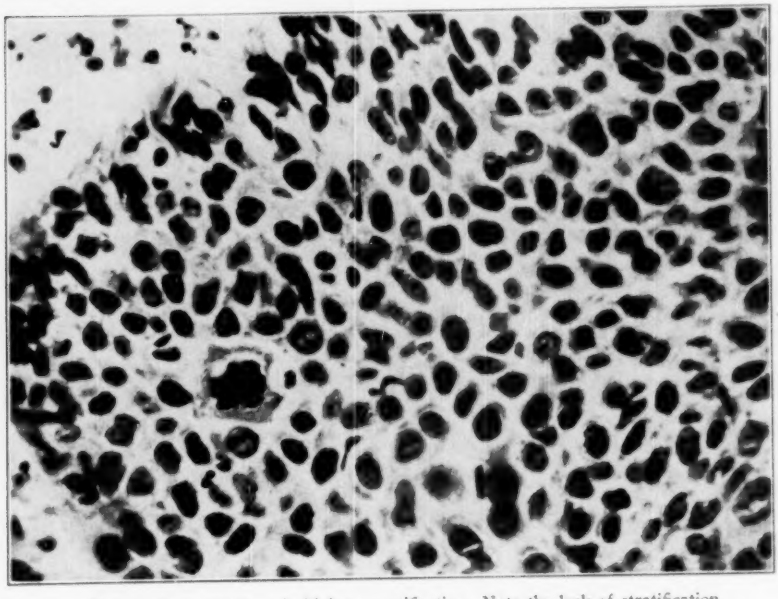

Fig. 5.—The carcinoma in higher magnification. Note the lack of stratification, the loss of polarity of cells and the variation in size and shape of the nuclei.

On May 15 a laryngectomy was performed. There were adhesions between the right thyroid cartilage, cricoid ring and thyroid gland. The thyroid gland was enlarged, and the arteries were thickened and rigid. The larynx was removed together with the first tracheal ring. Recovery was uneventful.

Macroscopic Examination.—The larynx was not symmetric because the left thyroid plate seemed to be wider than the right. Below the superior margin of the right thyroid plate was a defect in the cartilage the size of a pinhead, which led into the piriform sinus and which had apparently served as a passage for an anastomosing blood vessel. The right side of the cricothyroid membrane was covered with a thick layer of tissue which had firmly grown together with the cartilages as well as with the cricothyroid membrane. The upper part of the

cricoid plate was ossified. The prelaryngeal gland was enlarged. In the inside of the larynx there was no tumor (fig. 6). The posterior part of the left true cord was present and was continued by a thick scar, which took the place of the anterior commissure. The right true cord was entirely replaced by scar tissue. Below the scar tissue in the anterior commissure on the right side was a subepithelial hemorrhage, probably a result of the biopsy. Above the right "vocal cord" was a sausage-shaped thickening of the mucosa, which extended from the lateral margin of the epiglottis toward the center of the anterior commissure and was caused by an edema of the mucosa. The mucosa of the subglottic area was apparently under extreme tension and formed folds which converged toward the center of the anterior commissure. The mucosa seemed to be slightly thickened and showed numerous grooves the size of a pinpoint, which apparently were openings of glandular ducts. At the mesial surface of the right arytenoid cartilage, the mucosa had the appearance of leukoplakia. There was no hyperkeratosis or papilloma.

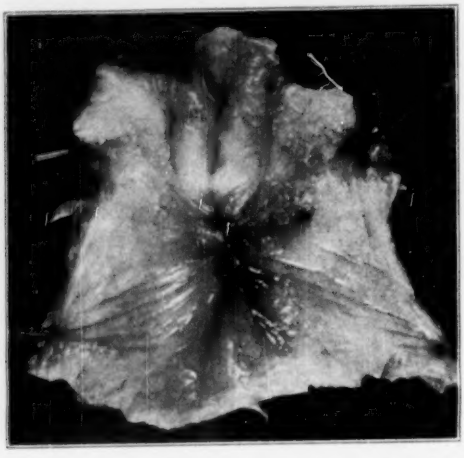

Fig. 6.—Laryngeal mucosa removed from the laryngeal cartilages. Note the absence of the tumor, formation of folds, hemorrhage in the area of the anterior commissure, edema of the epiglottis and the remnant of the left vocal cord.

The mucosa of the larynx was carefully removed from the cartilages, and interrupted serial sections were prepared. After removal of the mucosa the lateral cricoarytenoid muscle on the left side seemed to be normal. On the right side it was replaced by firm connective tissue, which was a continuation of the connective tissue at the outside of the larynx.

Microscopic Examination.—In the area of the vocal cords, the anteroposterior diameter of the larynx was slightly shortened (fig. 7). The mucosa consisted of connective tissue, glands, muscles and epithelium. The anterior wall and the anterior part of both lateral walls consisted of moderately inflamed connective tissue and of granulation tissue rich in blood vessels. In the scar, filling the incision of the laryngofissure, the connective tissue fibers ran in all directions and showed, to a small extent, hyaline degeneration. The connective tissue

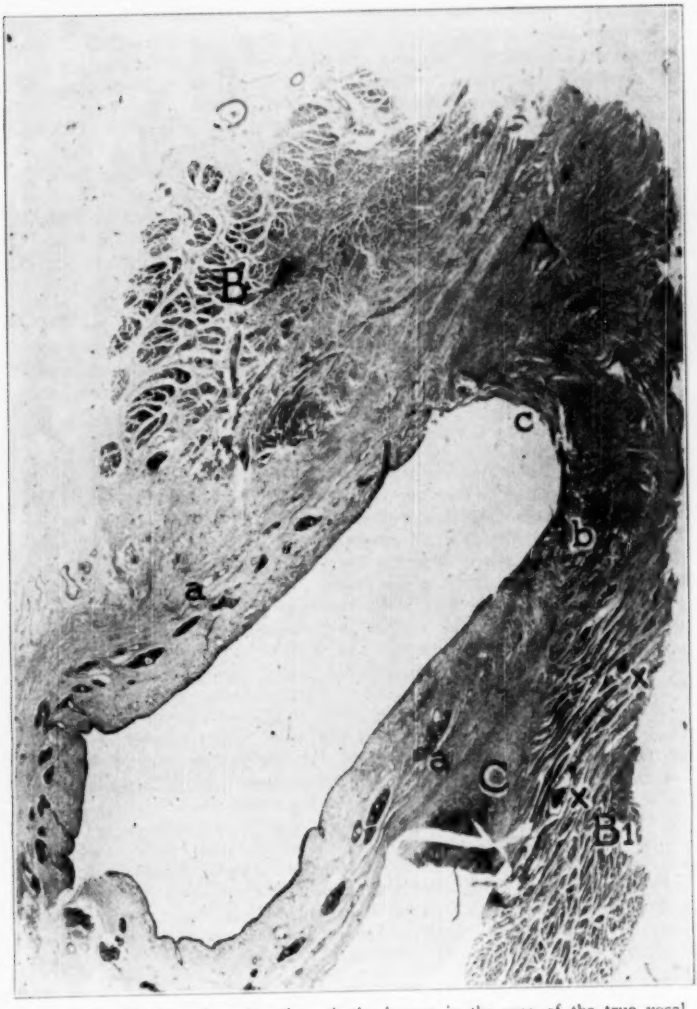

Fig. 7.—Horizontal section through the larynx in the area of the true vocal cords; (*A*) connective tissue scar in the area of the anterior commissure; (*B*) vocal muscle on the left side; (B_1) lateral cricoarytenoid muscle plus remnants of vocal muscle on the right side, containing trichinellas (*x*); (*C*) vocal process of the right arytenoid cartilage; (*a*) glands; (*b*) acute hemorrhagic inflammation. At the anterior wall of the glottis (*c*) the epithelium is absent. The other walls of the glottis show partly respiratory epithelium, partly low squamous epithelium and partly regenerated epithelium (no tumor).

scar extended from the midline toward the right side. Within the connective tissue were narrow channels filled with granulation tissue which contained nodules. The nodules consisted of a necrotic center surrounded by giant cells, lymphocytes and connective tissue cells. The remnants of the vocal muscles extended into the connective tissue. There were more muscle fibers on the left side than on the right. The muscle fibers on the right side were atrophic and poor in nuclei. On both sides there were many trichinellas within the muscles. The left vocal muscle extended to the midline, but some fibers crossed the midline. The fibers ran from the posterior wall to the anterior wall of the larynx, and some fibers ran from the lateral wall toward the midline. The right vocal muscle did not reach the midline; the fibers ran exclusively from the posterior wall toward the anterior wall of the larynx. It appeared that the scar within the area of the laryngofissure had pulled the left vocal muscle beyond the midline.

Glands were found within the posterior wall and the posterior parts of the lateral walls of the larynx (fig. 7). They showed normal activity but only a small number of lymphoid cells. On the left side the glands extended toward the anterior wall, but at that site they were atrophic. There was no elastic tissue in the mucosa. There was an acute hemorrhagic inflammation, which was more evident on the right side than on the left.

The epithelium was absent on the anterior wall and on the anterior parts of the lateral walls (fig. 7). It was doubtful whether or not this absence was caused by the inflammation or by the biopsy—more likely the biopsy. In the other parts of the larynx the epithelium consisted of one or two layers of flat cells which failed to form an actual epithelium. They were arranged after the pattern of mesothelium. Toward the posterior wall was a stratified squamous epithelium, which changed considerably and abruptly in thickness. There was no derangement of the epithelial structure. Respiratory epithelium was absent.

In the area of the false cords the glottis became longer in the anteroposterior diameter. Toward the anterior wall the glottis formed an acute angle and showed numerous crypts extending to the right and the left. On the anterior wall and the anterior parts of the lateral walls there was normal respiratory epithelium. The rest of the mucosa of the larynx was covered with stratified squamous epithelium, variable in thickness and exhibiting parakeratosis on the left side. The respiratory epithelium toward the epiglottis expanded; only the posterior wall was covered with a thick squamous epithelium with low papillae. The inflammatory infiltration was less evident, but there was a pronounced inflammatory edema of the anterior wall and of the anterior parts of the lateral walls.

In the subglottic space the larynx showed the same configuration as in the area of the vocal cords. The anterior wall and the anterior parts of the lateral walls were covered with a low epithelium, consisting of one layer of cubical cells or of one to three layers of squamous cells. On the left side were small spots of respiratory epithelium. Toward the trachea the spots of respiratory epithelium became enlarged on the anterior, left lateral and posterior walls. Between the spots there was transitional or low epithelium of one layer of cuboid cells or two layers of flat cells. The mucosa was edematous on both sides, more on the right than on the left. On the right side were many dilated capillaries and recent hemorrhages. Because of edema the mucosa formed small polyps bulging into the lumen and partly covered with epithelium. There were dilated and empty arteries and veins in the lateral wall, particularly in the anterior part of the left side, which ran in the mucosa from above downward.

Closer to the trachea the respiratory epithelium expanded rapidly and occupied all walls except the right lateral wall, which was covered with transitional

epithelium or with stratified squamous epithelium. The inflammatory edema on the right side continued. The glands on the right side were atrophic to a great extent. Finally, the entire mucosa was covered with respiratory epithelium. Below the epithelium were a small number of lymphocytes. Only the glands on the left side showed a normal number of lymphocytes. Close to the trachea glands were found everywhere, including the anterior wall. They were normal. The inflammatory edema and the subepithelial hemorrhages on the right side extended down toward the trachea.

The posterior cricoarytenoid muscles contained a large number of trichinellas, but there was no difference between the muscles on either side.

COMMENT

This case report was one of papillary carcinoma of the larynx associated with trichinosis, which had caused persistent hoarseness for only four months. At the first examination of the patient there was evident only a thickening and a yellow discoloration of the anterior portion of the right vocal cord associated with multiple minute epithelial defects. An attempt was made to remove the carcinoma by laryngofissure. At operation the carcinoma was found to extend into the subglottic space. Since the patient had not given his consent for a laryngectomy, I proceeded to complete the laryngofissure, having in mind the experience of Sir St. Clair Thomson,[1] who achieved a permanent cure in 6 of 13 cases of subglottic carcinoma by performing a laryngofissure. He stated that even a bilateral subglottic carcinoma, if it has not invaded the actual anterior commissure, may eventually be treated by laryngofissure. In the presented case the right true cord, part of the left true cord, part of the false cords on both sides and a considerable amount of the subglottic mucosa on the right side were removed.

Microscopic examination revealed a tumor which at several sites had the structure of a simple papilloma; at other sites the wild growth of epithelial cells, the abnormal and variable shape of the nuclei and the loss of stratification of the epithelium indicated a carcinoma. Below the edge of the true cord the tumor consisted of an accumulation of pleomorphic cells, forming a nodule. Both the papillomatous and the carcinomatous part failed to extend deeply into the mucosa; they did not reach the vocal muscle, although the carcinomatous parts did extend deeper than the papillomatous parts. The lateral extension of the tumor was greater than the extension in depth. The tumor occupied the right subglottic area but did not reach the edge of the true cord. It also did not invade the actual anterior commissure but extended below the commissure from the right side to the left. The surgical excision of the tumor was apparently appropriate except at the inferior boundary of the tumor, where the excision was not made at a sufficient distance

1. Thomson, St. C.: Subglottic Carcinoma of the Larynx, J. Laryng. & Otol. **52**:803, 1937.

from the tumor, although in this area the tumor had distinctly papillomatous structure.

In addition, there was widespread trichinosis of the laryngeal muscles. A large amount of calcium was discovered between the fibers of the right vocal muscle. Many muscle fibers were degenerated; yet the motility of the larynx had been normal, probably because the inflammation caused by the trichinosis was slight.

Subsequent to the operation, granulations developed in the area of the false cords and of the anterior commissure, and they had to be removed twice. This development of the granulations was caused by a severe inflammation, which, despite the use of penicillin, continued for approximately three months. It is likely that the surgical removal of the granulations had encouraged the inflammation, which extended toward the aryepiglottic fold and through the cricothyroid membrane on the right side. At the time of laryngectomy the inflammation was still partly active; it had partly caused the formation of connective tissue within the larynx as well as on the outside of the larynx. On the outside a thick layer of connective tissue had formed and covered the cricothyroid membrane on the right side and extended through the membrane into the larynx, replacing the lateral cricoarytenoid muscle on the right side.

Despite the severe inflammation, the connective tissue scars have formed a new "vocal cord." Examination of the specimen proved that the connective tissue scar in the midline had exerted a pulling force on the rest of the mucosa, thus causing folds of the mucosa, which converged toward the scar in the incision of the laryngofissure. These folds served as vocal cords. The glottic chink was slightly shortened, but there was no formation of a neomembrane in the area of the anterior commissure, as Fraenkel[2] noticed in one specimen four months after chordectomy.

The epithelium had not been restored within approximately three months. It is true that the mucosa of the entire larynx was covered with cells, but at the site of the operation there were only one to three layers of flat or cuboid cells, which were arranged more after the pattern of mesothelium than epithelium. In contradistinction, Citelli,[3] who studied the regeneration of the vocal cords after chordectomy in dogs, stated that eighteen to twenty days after the operation there are one to two layers of squamous cells covering the postoperative granulation tissue; in the next days more layers of squamous epithelium are added until, at forty-five days after the operation, the normal epi-

2. Fraenkel, B.: Zur Regeneration exstirpierter Stimmlippen, Arch. f. Laryngol. u. Rhinol. **20**:98, 1907.

3. Citelli: Chordectomia externa und Regeneration der Stimmlippen, Arch. f. Laryngol. u. Rhinol. **20**:73, 1907.

thelium has been restored. The delay of the epithelial regeneration in the presented case was obviously caused by the age and the original disease of the patient.

The part of the larynx which had not been involved by the operation had a normal epithelium, except the posterior wall where the squamous epithelium displayed considerable and abrupt changes in thickness without derangement of structure. On no place was there a sign indicating the formation of a carcinoma or even of a papilloma. The absence of such means that the excision performed for biopsy had removed the entire tumor. In other words, this means that there was no indication for performing a laryngectomy. Naturally, this raises the question of what type of procedure should be applied in instances of superficial epidermoid carcinoma of the larynx.

In laryngology, this problem has not been given much attention, although the relation between pachyderma and papilloma of the larynx, on the one hand, and carcinoma, on the other, has been frequently studied. However, not much information is available concerning the relation between carcinoma in situ and frank laryngeal carcinoma. In this respect, the most exhaustive study was presented by Brighton and Altmann,[4] who reported 3 cases of superficial lesions of the larynx:

In the first case, that of a man aged 70, there was a small tumor on the right vocal cord, which was removed with the biting forceps. The lesion was a papillary growth with extreme hyperplasia, considerable variation in morphologic structure of the cells and many mitotic figures. One year later the tumor recurred at exactly the same place from which the first growth had been removed. Now the diagnosis of Bowen's disease was made, based on the finding of poikilokaryosis (variation of cells in size and shape), Bowen cells (monster cells with several nuclei centrally clumped together), Darier cells (cells with a clear vacuole partly or completely surrounding the nucleus) and lack of infiltrative growth. Seven months later there was a second recurrence at the same site. Again the diagnosis was Bowen's disease. There were no other recurrences in the next fourteen months.

In the second case, that of a man aged 58, there was a small pale mass with a smooth surface on the right vocal cord near the anterior commissure, which was removed with biting forceps. The diagnosis was made of a superficial squamous cell epithelioma akin to Bowen's disease (of bowenoid type). In the next seven months there was no recurrence after the excision of the original tumor.

In the third case, that of a woman aged 59, a biopsy specimen was taken from the left vocal cord and the arytenoid cartilage. The diagnosis was made of superficial squamous cell epithelioma (bowenoid type). After a few days a laryngofissure was performed, and the left vocal cord, the ventricular band and the mucous membrane covering the left arytenoid region were removed. The previous diagnosis was confirmed. There was no recurrence in twenty months.

4. Brighton, G. R., and Altmann, F.: Bowen's Disease and Superficial Mucosal Epithelioma of Upper Respiratory and Alimentary Tracts, Arch. Otolaryng. **36**:354 (Sept.) 1942.

Brighton and Altmann stated that there is a gradual, but not an essential, histologic difference between typical Bowen's disease, frank cancer with Bowen's lesions at the periphery, lesions with some characteristics of Bowen's disease and lesions with the same type of intraepithelial spread but without the cellular characteristics of Bowen's disease. In all these diseases the intraepithelial spread is the most important feature. So far as the treatment is concerned, Brighton and Altmann are apparently in favor of a radical excision by laryngofissure in instances of superficial carcinoma of the larynx. In the case of Clerf,[5] the diagnosis of an invasive type of epithelial tumor was made in 1927 and a laryngectomy was advised. The patient refused, and the laryngectomy was performed in 1938. An ulcerative papillary tumor was discovered, involving the entire left vocal cord, extending into the ventricle and involving the anterior extremity of the left ventricular band. It also extended across the midline, involving the anterior third of the right vocal cord. There was pronounced subglottic extension. Clerf stated that it was possible that there actually had been a carcinoma in situ in 1927.

In the case of Snitman[6] there was a small warty growth on the anterior half of the left vocal cord. At laryngofissure 0.5 cm. of the anterior portion of the right true and false cords was removed along with the complete left cord, including the vocal process of the arytenoid cartilage. An extensive squamous cell carcinoma was found, which apparently was not radically removed. Approximately eight months later a web formation was discovered at the anterior commissure. Biopsy specimens taken from the web and the anterior third of the right true cord revealed carcinoma in situ and a few areas of squamous cell carcinoma. Snitman stated the opinion that the carcinoma in situ is the residual malignant tissue subsequent to the incomplete excision of the original tumor.

Disregarding the strictly histologic aspect of the problem, the clinician is mainly interested in two questions: 1. Has it been proved that a superficial epidermoid carcinoma (including Bowen's disease) is invariably the primary phase of a frank clinical carcinoma? 2. Does the finding microscopically of a superficial epidermoid carcinoma warrant the radical operation on the organ involved? These questions, of course, deal exclusively with instances in which the superficial carcinoma is the sole pathologic finding, viz., in which it is not associated with a frank carcinoma.

5. Clerf, L. H.: Cancer of the Larynx: Report of Unusual Case, Tr., Am. Laryng. A. **66**:167, 1944.

6. Snitman, M. F.: Carcinoma of the Larynx, Arch. Otolaryng. **42**:178 (Sept.) 1945.

The experience of the laryngologists mentioned here is apparently not sufficient to answer these questions definitely. Unfortunately, the reports of the gynecologists who have studied this problem for a long time also do not furnish a final answer. Schiller [7] deserves the chief credit for recognizing the importance of the preinvasive type of carcinoma as the forerunner of those tumors which show similar intraepithelial changes plus histologic evidence of invasion. Schiller expressed the opinion that such a lesion was not precancerous but an early phase of carcinoma. This doctrine has caused a great deal of discussion, mainly among the gynecologists. At present, the view seems to be prevalent that superficial carcinoma (or preinvasive carcinoma, or intraepithelial carcinoma or carcinoma in situ) is the primary phase of frank carcinoma but that there are exceptions. Novak [8] maintained that the total number of cases available in the literature in which genuine invasive carcinoma has developed from superficial carcinoma is still extremely rare (1947). In addition, the duration of the preinvasive phase is extremely variable. Pund and Auerbach [9] asserted that invasion begins about six years later than the preinvasive phase and that frank carcinoma may be delayed as long as an additional six years before ulceration is obvious.

The relation between superficial carcinoma and invasive carcinoma not being definitely established, the treatment in cases of superficial carcinoma is likewise not standardized. One group of gynecologists (Schiller,[7] Pund and Auerbach [9]) suggested total hysterectomy; another group (Schmitz and Benjamin [10]), cervix amputation, and a third group (TeLinde and Galvin,[11] Novak [8]), simple excision in instances of superficial carcinoma of the cervix.

This brief summary of the present viewpoints is not very enlightening. From the practical point of view there is not much gained from the concept of carcinoma in situ, because apparently the progress from preinvasive carcinoma to invasive carcinoma is not less uncertain than the progress of so-called precancerous lesions to actual cancer. In the presented case the carcinoma in situ appeared on the place where a papillary carcinoma had been removed previously. It is not likely that it was a remnant of the papillary carcinoma, similar to that in the case of Snitman,[6] because the microscopic examination proved that the

7. Schiller, W.: Ueber Fruehstadien des Portiocarcinoms und ihre Diagnose, Arch. f. Gynäk. **133**:211, 1928.

8. Novak, E.: Cancer of the Uterus, J. A. M. A. **135**:199 (Sept. 27) 1947.

9. Pund, E. R., and Auerbach, S. H.: Preinvasive Carcinoma of the Cervix Uteri, J. A. M. A. **131**:960 (July 20) 1946.

10. Schmitz, H., and Benjamin, E. L.: The Early Histologic Diagnosis of Carcinoma of the Uterine Cervix, J. A. M. A. **103**:808 (Sept. 15) 1934.

11. TeLinde, R. W., and Galvin, G.: The Minimal Histological Changes in Biopsies to Justify the Diagnosis of Cervical Cancer, Am. J. Obst. & Gynec. **48**:774, 1944.

excision of the original carcinoma was complete, although at the inferior boundary of the tumor not sufficient healthy tissue was excised along with the tumor. In addition, two biopsy specimens which were taken from the same area did not show carcinoma. I believe that the carcinoma in situ of the presented case developed in the process of regeneration within two months. However, more important is the fact that the carcinoma in situ was, according to the microscopic examination, no indication for performing a laryngectomy at the time at which it was performed. There was likewise no indication prophylactically for performing a laryngectomy, because, according to present knowledge, it was not possible to predict if and when the carcinoma in situ would turn into a frank carcinoma. And even if one adheres to the view of Pund and Auerbach,[9] who stated that invasion begins about six years later than the preinvasive phase, it must be kept in mind that this statement concerns superficial carcinoma of the cervix, which is supposed to appear in women at the average age of 36.6 years. The age of the patient in the presented case was 62, which makes a difference so far as the speed of tumor growth is concerned, particularly in a case in which the lymph vessels had been partially obliterated by a previous laryngofissure.

Disregarding these considerations, the laryngectomy was performed because the diagnosis of superficial carcinoma was quite alarming and was strictly overestimated. One could of course argue that in due time this particular patient would have required a laryngectomy because the excision of the original tumor had not been radical enough. But this is not the point in question. The fact is that the diagnosis of superficial carcinoma had served as an indication for laryngectomy, and this was evidently not correct.

From this experience the following conclusion may be drawn: Superficial carcinoma of the larynx may be an actual carcinoma microscopically; it is not a genuine carcinoma from the therapeutic point of view. For this reason there is primarily no indication to perform a laryngofissure and certainly not a laryngectomy; the carcinoma in situ may be removed by the endolaryngeal route if it forms a localized tumor, and further steps may be taken if this procedure does not prove to be effective.

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PALATAL MYOCLONUS

Report of Three Cases

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PALATAL myoclonus is a neurologic symptom complex, which apparently occurs with sufficient frequency to be of interest to the otolaryngologist. This obscure phenomenon consists in clonic contractions of one or more of the muscles attached to the eustachian tube and soft palate and a synchronous ticking noise easily audible to both patient and observer. The contractions are not rhythmic, as a rule, and their rate varies in individual cases from as few as 80 to 140 or 150 per minute. They may appear either as a small dimpling of the soft palate or as a complete elevation of one or both palatal arches and uvula. The tinnitus is best described as a ticking or clicking noise and can be simulated by the snapping of one finger nail over another. The sound may be heard at distances varying from that of direct contact with the patient's ear to several feet. Nasopharyngoscopic examination in these cases will reveal a contraction of the tubopalatal muscles, which exert a downward pull on the eustachian tube and an upward pull on the soft palate. It is stated by the majority of those who have reported this disease that the clonic contractions of the tubal muscles cause a sudden separation of the moist walls of the tube, thus producing the tinnitus. Several authorities have been of the opinion, however, that the auditory muscles, the stapedius and tensor tympani, can produce an audible click by their sudden contraction.

The muscles attached to the cartilaginous portion of the eustachian tube are as follows:

1. the levator veli palatini, some fibers of which arise from the inferior cartilaginous portion of the eustachian tube and are inserted into the aponeurosis of the soft palate;

2. the tensor veli palatini, one root of which arises from the lateral cartilaginous lamina and from the fibrous portion of the tube and is inserted into the posterior border of the hard palate and into the aponeurosis of the soft palate;

3. the salpingopharyngeus, which arises from the lower posterior part of the tube and joins the fibers of the pharyngopalatinus, which becomes a part of the soft palate.

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Candidate's thesis presented in partial fulfilment of the requirements for membership in the American Laryngological, Rhinological and Otological Society.

Contraction of the salpingopharyngeus draws the medial lamina of the eustachian tube downward and backward, while the tensor veli palatini pulls the lateral wall anterolaterally, thus separating it from the posterior wall. It is quite obvious that a contraction such as that seen in palatal myoclonus will cause a simultaneous opening of the eustachian tube and a dimpling of the soft palate or, as in some cases, an elevation of the entire palate. The dimpling appears to occur when only a few fibers are contracting, exerting a pull on the soft palate where it is attached to the posterior border of the hard palate. Sometimes the muscles of the pharynx and larynx are involved, as seen by mirror examination. There are reports of extensive myoclonias in the neurologic literature, in which the usual syndrome is augmented by contractions of the mimetic and ocular muscles, the diaphragm and muscles of the extremities. But in the majority of these cases no mention is made of an audible tinnitus. It is quite possible, therefore, that the presence of tinnitus is limited to the relatively mild myoclonias, in which only the tubopalatal muscles are involved.

Iglauer [1] and other investigators reported another type of objective tinnitus, namely, that caused by anomalies of the blood vessels in or about the ear. This vascular tinnitus can be easily differentiated, however, since it is usually a soft blowing murmur synchronous with the pulse. Furthermore, pressure over the carotid artery will cause a diminution or disappearance of the sound.

Most of the reports in the otolaryngologic literature are based on the clinical observation of cases; the majority of these cases were mild, and the ages of the patients ranged from 9 to 63 years. Considerable work of an investigative nature into the pathologic basis of the myoclonias has been done by many neurologists. It was the interesting thesis of Gallet,[2] student of Foix [3] which focused attention in 1927 on the subject of palatal myoclonus. Gallet surveyed 37 cases reported up to the time of his writing. Subsequent to his report many cases have been added to the neurologic literature, in some of which autopsy was eventually performed. Leshin and Stone [4] reported a case in 1931, and Walter Freeman [5] added 2 cases in 1933, in which autopsy was eventually made. Guillain and Mollaret [6] reported 2 cases in 1935.

1. Iglauer, S.: Objective Tinnitus Aurium: Report of Four Cases, Tr. Am. Laryng. A. **39**:401, 1933.

2. Gallet, J.: Le nystagmus du voile (myoclonie vélo-pharyngo-laryngée) et les myoclonies associées oculaires, faciales, sus-hyoïdiennes, diaphragmatiques, Poitiers, Soc. fr. impr., 1927, p. 139.

3. Foix, C., and Hillemand, P.: Contribution à l'étude des ramollissements protubérantiels, Rev. de méd. **43**:287, 1926.

4. Leshin, N., and Stone, T. T.: Continuous Rhythmic Movements of the Palate, Pharynx and Larynx, Arch. Neurol. & Psychiat. **26**:1236 (Dec.) 1931.

5. Freeman, W.: Palatal Myoclonus: Report of Two Cases with Necropsy, Arch. Neurol. & Psychiat. **29**:742 (April) 1933.

6. Guillain, G., and Mollaret, P.: Le syndrome myoclonique synchrone et rythmé vélo-pharyngo-laryngo-oculo-diaphragmatique, Presse méd. **43**:57, 1935.

Davison, Riley and Brock [7] in 1936 added another case in which autopsy was eventually performed and reviewed all of the cases with autopsy up to that time. Sittig and Haskovec [8] reported 4 cases in 1939, in 1 of which autopsy was eventually made. Murphy and Langworthy [9] added 2 cases with autopsy in 1939. Dobson and Riley [10] added 6 cases of rhythmic myoclonus in 1941, in none of which autopsy was performed.

The majority of the patients were over 50, with various degrees of pathologic change of the central nervous system, frequently as a result of cerebral arteriosclerosis. Davison, Riley and Brock, after a careful analysis of the histopathologic picture in the cases in which autopsy was eventually performed, reached the following conclusion:

> From a study of this instance [their case report] and those previously reported, it may be concluded that the lesion which permits the appearance of these myoclonic movements is usually confined to the ipsilateral central tegmental tract and the ipsilateral inferior olivary nucleus. In a few cases, the lesions were situated in the dentate nucleus and contralateral inferior olivary nuclei.[7]

A résumé of the myoclonic movements observed in the cases surveyed by Davison, Riley and Brock [7] follows:

Foix, case 1: facial myoclonic movements on both sides

case 2: left-sided facial, palatal and laryngeal myoclonic movements

case 3: bilateral palatal myoclonic movements

case 4: palatopharyngolaryngeal myoclonic movements, more pronounced on the left

Van Bogaert: Right-sided myoclonic movements involving the face, eyelid, chin and platysma muscle (the palate was spared)

Van Bogaert and Bertrand: Right-sided palatopharyngeal and diaphragmatic myoclonic movements

Lhermitte, Levy and Trelles: Bilateral palatopharyngolaryngeal myoclonic movements, more pronounced on the left

Freeman, case 1: Left-sided palatopharyngeal myoclonic movements

case 2: Bilateral palatopharyngeal myoclonic movements, more pronounced on the left and slight on the right

Shugrue: Right-sided palatopharyngeal myoclonic movements

Guillain, Mollaret and Bertrand: Bilateral palatopharyngolaryngodiaphragmatic myoclonic movements

Guillain, Thurel and Bertrand: Bilateral palatopharyngo-oculodiaphragmatic myoclonic movements, more pronounced on the left

Garcin, Bertrand and Frumason: Palatal myoclonic movements

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7. Davison, C.; Riley, H. A., and Brock, S.: Rhythmic Palatal Myoclonus of Muscles of Palate, Larynx and Other Regions, Bull. Neurol. Inst. New York **5**:94, 1936.

8. Sittig, O., and Haskovec, V.: Palatal Myoclonus, Arch. Neurol. & Psychiat. **42**:413 (Sept.) 1939.

9. Murphy, E. L., and Langworthy, O. R.: Palatal Myoclonus: A Clinical and Anatomical Report of Two Cases, South. M. J. **32**:1035, 1939.

10. Dobson, J. P., and Riley, H. A.: Rhythmic Myoclonus: A Clinical Report of Six Cases, Arch. Neurol. & Psychiat. **45**:145 (Jan.) 1941.

Lhermitte, Levy and Trelles: Left-sided palatopharyngo-ocular myoclonic movements.

Davison, Riley and Brock: Bilateral ocular, palatal, oral, pharyngeal and laryngeal myoclonic movements, more pronounced on the left.

As is evident from the summary of these cases, the myoclonus may be either unilateral or bilateral and may involve the palatopharyngolaryngeal muscles and sometimes the diaphragm, ocular muscles and muscles of the face and the platysma; and, as in the case of Van Bogaert, it may completely spare the palatal muscles. Freeman mentioned the presence of tinnitus in palatal myoclonus as part of the syndrome but did not specify whether it was present in his 2 cases. As a matter of fact, in very few of the cases reported in the neurologic literature is any specific reference made to the presence of audible tinnitus. On the other hand, practically every case reported in the otolaryngologic literature features the objective tinnitus. These cases are referred to by their authors simply as "objective tinnitus of muscular origin" to differentiate them from those of vascular origin. In practically every instance, however, the presence of a synchronous contraction of the tubopalatal muscles is carefully described, thus presenting the typical syndrome of palatal myoclonus.

One of the earliest reports by an otolaryngologist in this country is that of Wells,[11] who presented his observations on 2 cases of objective tinnitus. These had been previously reported by him at the meeting of the Section on Otolaryngology of the American Medical Association in June 1904. He stated that in all the literature 17 cases had been previously reported on this particular type of tinnitus. He described minutely his observations on the contractions of the tubal muscles, which in his opinion caused the objective tinnitus. Wells credited Johannes Müller (1837) as the first one to recognize the muscular origin of certain objective sounds in the ear. Müller believed that they were caused entirely by contraction of the tensor tympani muscle. Wells also stated that Beck in 1867 was the first to prove by posterior rhinoscopy that the cause of the objective tinnitus was contraction of the muscles about the eustachian tube, a theory previously advanced by Politzer.

On the other hand, Kafka[12] stated the opinion that this sound was produced by sudden contraction of the stapedius or the tensor tympani muscle.

Jones and Knudsen[13] in 1928 reported a case of objective tinnitus associated with spasmodic contractions of the palatal muscles. They

11. Wells, W. A.: Two Cases of Objective Aural Tinnitus Due to Action of Tubo-Palatal Muscles, J. A. M. A. **44**:180 (Jan. 21) 1905.

12. Kafka, M. M.: Tinnitus Aurium: Etiology, Differential Diagnosis, Treatment and Review of Twenty-Five Cases, Laryngoscope **44**:515, 1934.

13. Jones, I. H., and Knudsen, V. O.: Certain Aspects of Tinnitus, Particularly the Treatment, Laryngoscope **38**:597, 1928.

stated that the sound was produced by the sudden opening of the eustachian tube with each contraction and called the condition pseudotinnitus.

Jennings [14] reported in 1938 the case of a child of 13 with an audible clicking noise in the ears present ever since the child could remember. There was a synchronous contraction of the palate, and the rate of contraction was 130 per minute.

Bredlau [15] in 1938 reported the case of a boy of 9 years in whom, during the course of a purulent otitis media, a clicking noise developed in the right ear that was easily audible to the examiner. The rate of the tinnitus was about 100 per minute, and there occurred occasional synchronous contractions of the palate on that side. Although the therapeutic measures instituted cured the otitis media, the ticking sound persisted. Bredlau [15] quoted Friedmann, who reported a similar case in a 9 year old girl; in this case there were rhythmic movements of the soft palate synchronous with the noise, and this was ascribed to the contractions of the tubal muscles.

Young [16] in 1942 reported the case of a man of 29 who had a clicking noise in the right ear that could be heard 4 feet (1.22 meters) away. Later the left ear was similarly affected, though not as intensely. The contractions were at the rate of about 84 per minute, irregular and not synchronous with the pulse. There was no evidence in this case of myoclonic movements around the eustachian tube, pharynx or palate.

In 1947 Davis and Kirchick [17] reported a case before the Academy of Otolaryngology of a patient with an audible clicking noise of several months' duration. This man had rhythmic myoclonus of the palate, synchronous with a ticking noise in his right ear. He had had hypertension and had been subjected to considerable emotional strain due to financial setbacks. The symptoms began after his ears had been plugged by forceful blowing of the nose. Nasopharyngoscopic examination disclosed contraction of the muscles about the tube, but both the contractions and the noise could be suppressed by the patient if he held his nose and plugged his ear with his finger. This is the only case in which such a phenomenon has been reported. Therapy in this case included injections of procaine hydrochloride (novocain®) into the tubopalatal muscles, plus injections of calcium gluconate, thiamine hydrochloride and neostigmine (prostigmin®), none of which gave any relief. Curare, on the other hand, when injected into the tubal muscles caused a slowing of the contractions and the tinnitus for about one week. A neurologic consultant expressed the opinion that softening of the brain stem

14. Jennings, J. L.: Case Report of Unusual Noises of the Ear, Bull. Fulton Co. M. Soc. **12**:67, 1938.

15. Bredlau, E. A.: Objective Tinnitus Aurium, Arch. Otolaryng. **28**:193 (Aug.) 1938.

16. Young, N.: Objective Tinnitus, Brit. M. J. **1**:414, 1942.

17. Davis, D., and Kirchick, J. A.: Palatal Myoclonus, read before the Academy of Ophthalmology and Otolaryngology, Chicago, October, 1947.

had probably resulted in interference with the thalamo-olivary tract. This case report was illustrated with a moving picture of the muscular contractions and a recording of the tinnitus.

Pearson and Barnes [18] reported the case of a lieutenant in the Marine Corps, in whom objective tinnitus developed after the explosion of a 5 inch (12.7 cm.) gun. The patient was successfully treated by hypnosis, complete cessation of the tinnitus resulting. The authors reported an additional case of objective tinnitus in which a cure by hypnosis was likewise obtained. These 2 cases are the only ones I have found in the literature that report successful treatment by any method.

McGriff [19] reported the case of a Marine aged 34 with a ticking noise in his left ear after a gun explosion. Associated with the ticking sound could be seen a synchronous contraction of the tubopalatal muscles, as observed with the nasopharyngoscope. All local treatment attempted in this case failed. Although hypnosis was successfully induced, no improvement could be obtained with this therapy either.

REPORT OF CASES

CASE 1.—V. B., a farmhand aged 20, was seen in March 1935. He complained of a peculiar ticking noise, which could be heard by other people if they got close enough to his ears. This symptom had been present for about eight or ten years. The only illness that he could recall was herpes zoster at the age of 8, involving the thoracic region. An ear, nose and throat examination revealed no abnormalities except for an audible ticking noise, which could be heard by direct contact with the ear or by auscultation. The rate of ticking varied from 80 to 90 per minute, was not rhythmic and was not synchronous with the pulse. Examination of the palate showed a dimpling on each side of the palate when the mouth was stretched open; but when it was relaxed there was a definite upward pull on the uvula, which was synchronous with the ticking noise. Examination of the larynx disclosed no evidence of contraction of any of the laryngeal muscles. There were no contractions of the facial, diaphragmatic or thoracic muscles. Hearing was found entirely normal on audiometric examination. A slight nystagmus could be observed when the eyes were directed to either side. Response to caloric stimulation was somewhat delayed on the right side but normal in every other respect. Neurologic examination indicated no other pathologic signs; however, in view of the neurologic consultant's previous experience with 2 cases in which autopsy was eventually performed, he stressed the possibility of a lesion of the brain stem. It is regrettable that efforts to communicate with this patient have been unsuccessful, permitting no follow-up of the case.

CASE 2.—E. T., a Negro aged 63, was seen in November 1947. His chief complaint was a ticking sound in the left ear. The patient stated that in 1916 he had some dental work done and that about 1944 further work was done to repair a loose bridge. He noticed the ticking noise in the left ear several months after this dental work. It has persisted for the past three years. The only other complaint

18. Pearson, M., and Barnes, L. J.: Objective Tinnitus Aurium: Report of a Case with Recovery After Hypnosis, Arch. Neurol. & Psychiat. **59**:265 (Feb.) 1948.

19. McGriff, J. R.: Palatal Myoclonus, Staff Conference at Episcopal Eye and Ear Hospital, Washington, D. C., 1947.

which the patient had at this time was recurring pain over the left antrum. Examination showed no abnormalities of the ears, nose or throat. Roentgenograms of the sinuses and mastoid cells disclosed nothing abnormal. An audiogram showed practically normal hearing for a man of his age. There was no evidence of spontaneous nystagmus, past pointing or falling. The corneal reflex was normal and there were no ataxias or tremors. Caloric stimulation produced entirely normal responses. In a previous physical examination at another hospital a moderate degree of hypertension was found. The ticking noise in the ear could be heard at 3 to 4 feet (0.9 to 1.22 meters) on the left side but could not be heard on the right, nor was the patient aware of any noise in his right ear. Nasopharyngoscopic examination, however, revealed a contraction of the tubal muscles on both sides, much more pronounced on the left; there was a synchronous dimpling on both sides of the palate at the point of attachment of the soft palate to the hard palate and no evidence of myoclonic contractions of the laryngeal or any other muscles. The rate of ticking and contractions was 106 per minute and very irregular. Topical injections of tetracaine and procaine into the tubal muscles did not alter the contractions or the ticking noise.

Although this patient submitted willingly, it was impossible to achieve satisfactory hypnosis even after strong sedation was used. He seemed amused at the interest displayed in his case, particularly since he had learned to live with the tinnitus and suffered no discomfort except for the pain in the left antrum, which has not been satisfactorily explained.

Case 3.—H. G., a veteran aged 18, stated that in December 1946 while he was standing a few feet away from a 240 mm. gun it was fired without warning. He was knocked unconscious; he was put to bed and remained there for the rest of the day. The next day he felt quite well and had no ill effects. Shortly after this episode, he noticed an occasional ticking in his ears and a tendency toward frequent nosebleeds. The patient stated that at first he could control this ticking sound, but as time went on the ticking became continuous and uncontrollable. He was hospitalized at an Army hospital for several months, beginning in October 1947, where he received radiation therapy for lymphoid tissue in the nasopharynx. The rate of ticking has averaged 140 per minute. Peculiarly, the ticking may stop in one ear for as much as one hour at a time without stopping in the other. The family history revealed nothing significant except for the fact that one brother became hard of hearing after an accident in the service. Examination showed a slight deviation of the septum to the right; the sinuses and pharynx were normal. Both tympanic membranes appeared normal. An audiogram indicated some loss of hearing bilaterally, more pronounced in the lower frequencies but essentially good hearing in the conversational range. There was no evidence of spontaneous nystagmus, past pointing or falling. The corneal reflex was normal and there were no ataxias or tremors. A caloric test with water at 68 degrees F. elicited normal responses on both sides. Both eustachian tubes were entirely patent on inflation. Nasopharyngoscopic examination disclosed a contraction of the tubal muscles, exerting a somewhat downward pull on both eustachian tubes. There was a slight synchronous elevation of the soft palate on both sides, which could not be altered by topical application of tetracaine.

At one time during examination a motion of the right tympanic membrane was observed, consisting of an inward dimpling in the posterior inferior quadrant, lasting only about one minute. The patient was not aware of any sensation of motion within the ear during my period of observation. It is quite possible, therefore, that this phenomenon occurred without the patient's knowledge as part of his symptom

complex. The rate of contraction of the tympanic membrane was identical with that of the palate and eustachian tube and synchronous with the ticking noise. Permission could not be obtained from the patient and his widowed mother to attempt suggestion under hypnosis, because of their strong prejudice against this form of therapy.

COMMENT

These cases are all typical of those previously reported in the otolaryngologic literature as "objective tinnitus of muscular origin." There are individual variations in the symptom complex of this disease, but the most constant characteristic is contraction of the tubopalatal muscles which result in (1) a visible dimpling or elevation of the soft palate and (2) a subjective and objective tinnitus, usually described as a clicking or ticking sound.

In case 1 the disease apparently existed since early childhood, and there was no indication in the patient's history of any appreciable change during the eight or ten years in which he had it. Except for slight hypoactivity of one labyrinth, there were no neurologic manifestations suggesting an intracranial lesion, which might account for the myoclonic syndrome. In case 2, because of the patient's age and the evidence of hypertension, there is a closer resemblance to the cases reported in the neurologic literature. Case 3 is similar to those reported by Pearson and Barnes and by McGriff in that the myoclonic symptoms appeared subsequent to heavy concussion following gun explosions. All of these patients were veterans, and the 2 cases reported by Pearson and Barnes were the only ones I have found in which a cure was obtained through hypnotic suggestion. In McGriff's case, hypnotic suggestion was induced, yet the symptoms persisted. Hypnosis was recommended and tried unsuccessfully in case 2. Case 3 would seem to offer a greater opportunity for testing the efficacy of this therapy, since it was almost identical with those cases with successful treatment by Pearson and Barnes. The failure of hypnosis in McGriff's case, on the other hand, raises the question of its value in every instance. If the fundamental pathologic process is degeneration of the brain stem, as autopsy seems to indicate, the efficacy of any therapy must be doubted. The question arises, however, whether a central lesion is always present in the development of palatal myoclonus. Can severe shock induce purely functional reactions with the resultant muscular spasm of the tubopalatal muscles? Certainly, some of the case histories are suggestive of the latter explanation. Were it not for the fact that at least 2 patients are reported as cured, palatal myoclonus as a disease entity would be of no more than academic interest to the otolaryngologist. The study of the neuropathology, as does indeed the question of therapy in palatal myoclonus, lies in the province of the neurologist and the psychiatrist. The otolaryngologist, however, must have a knowledge of this disease, as he will very likely be the first to be consulted because of the annoying tinnitus.

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HERPES ZOSTER OTICUS (RAMSAY HUNT'S SYNDROME)

Comments on an Article by Johnson and Zonderman

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THE CASE of herpes zoster oticus reported by Johnson and Zonderman[1] deserves special attention. The authors, after careful examination and observation of the case, revealed a series of unusual findings. Their records particularly concern items such as the sites of the herpetic eruptions, the primary site of the herpetic infection, the possible path of its extension and the high cerebrospinal lymphocyte count. In their attempt to classify the case, they faced some difficulties, particularly with regard to the site of the facial nerve lesion, which was thought to be above the geniculate ganglion, an unusual site in instances of geniculate herpes. This location was revealed by application of the topognostic method previously utilized in my studies of the site of the facial nerve lesion in cases of Ramsay Hunt's syndrome,[2] referred to in detail by Johnson and Zonderman. They finally recommended principles by which the clinical entity Ramsay Hunt's syndrome, or "geniculate zoster" should be determined.

In this regard, it seems, they were somewhat influenced by findings and statements presented in my previous article, unfortunately misinterpreted by them. Consequently, their conclusions appear questionable to me.

My intention here is to offer possible interpretations concerning some of the unusual features manifested by the case, to provide an analysis possibly helpful for an adequate classification and to clarify those points in my article which are apparently responsible for some questionable conclusions presented by Johnson and Zonderman. In this way future confusion in determining the clinical entity of Ramsay Hunt's syndrome may be prevented.

My discussion will include the following topics: (1) the site of the herpetic eruptions, (2) the primary site of the herpetic infection, (3) the anatomic factors involved in the path of extension toward the seventh nerve, (4) classification and (5) the criteria responsible for restriction of the term Ramsay Hunt's syndrome.

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1. Johnson, L., and Zonderman, B.: Herpes Zoster Oticus ("Ramsay Hunt Syndrome"): Report of a Case, Arch. Otolaryng. **48**:1 (July) 1948.

2. Tschiassny, K.: The Site of the Facial Nerve Lesion in Cases of Ramsay Hunt's Syndrome, Ann. Otol., Rhin. & Laryng. **55**:152, 1946.

THE SITE OF THE HERPETIC ERUPTIONS

The first peripheral eruption noticed in the case was mainly a "stomatitis" involving the right side of the tongue and fauces. Simultaneously an inflamed right ear canal with exudate and thickened drum membrane was noticed. In my opinion this "stomatitis" should be regarded as a herpetic manifestation. Hunt,[3] on the basis of comparative anatomic studies and of Cushing's clinical observations concerning residual crude sensations still present at the anterior two thirds of the tongue subsequent to gasserian ganglionectomy and on the basis of his own studies on herpes zoster of the tongue associated with ipsilateral facial paralysis, postulated the existence of a "visceral" distribution of the sensory portion of the seventh nerve by vestigial fibers. In Hunt's concept these filaments provide additional common sensation to the anterior two thirds of the tongue and oral surface of the palate. In his series of 87 recorded cases, there were only 5 in which a herpetic eruption was reported at this location with ipsilateral facial paralysis. In 1 case the glossopalatine changes were preceded by, in 1 case accompanied with, and in 3 cases not accompanied with, auricular herpes.[31]

It should be kept in mind that four different ganglions may be involved in lingual herpes zoster. They include the geniculate and the gasserian ganglion for the anterior two thirds of the tongue and the petrosal and the nodosal ganglion for the base of the tongue, the last-named ganglion involving only a wedge-shaped area bilaterally in front of the epiglottis.

In the present case the primary, mainly "visceral" geniculate herpes was followed after three weeks by a secondary "somatic" crop of lesions at the concha. This structure represents the center of the

3. Hunt, J. R.: (*a*) Herpetic Inflammation of the Geniculate Ganglion, J. Nerv. & Ment. Dis. **34**:73, 1907; (*b*) Herpetic Inflammation of the Geniculate Ganglion: A New Syndrome and Its Aural Complications, Arch. Otol. **36**:371, 1907; (*c*) Neuralgic Affections of the Geniculate Ganglion, J. Nerv. & Ment. Dis. **34**:21, 1907; (*d*) Otalgia Considered as an Affection of the Sensory System of the Seventh Cranial Nerve, Arch. Otol. **36**:543, 1907; (*e*) A Further Contribution to the Herpetic Inflammation of the Geniculate Ganglion, Am. J. M. Sc. **86**:226, 1908; (*f*) Ein Fall von Polyomyelitis posterior des Ganglion geniculi, Neurol. Centralbl. **27**:514, 1908; (*g*) The Sensory System of the Facial Nerve and Its Symptomatology, J. Nerv. & Ment. Dis. **36**:321, 1909; J. Ophth. & Otol. **4**:89, 1910; (*h*) Sensory Symptomatology and Syndrome of the Facial Nerve, J. Laryng. **25**:405, 1910; (*i*) The Symptom-Complex of Acute Posterior Poliomyelitis of the Geniculate, Auditory, Glossopharyngeal and Pneumogastric Ganglia, Arch. Int. Med. **5**:631 (June) 1910; (*j*) The Cutaneous Zone of the Facial Nerve, New York M. J. **102**:777, 1915; (*k*) The Sensory Field of the Facial Nerve (A Further Contribution to the Symptomatology of the Geniculate Ganglion), Brain **38**:418, 1915; J. Nerv. & Ment. Dis. **43**:156, 1916; (*l*) Geniculate Neuralgia (Neuralgia of the Nervus Facialis): A Further Contribution to the Sensory System of the Facial Nerve and Its Neuralgic Conditions, Arch. Neurol. & Psychiat. **37**:253 (Feb.) 1937.

"geniculate zone" at the external ear. This area was mapped out by Ramsay Hunt and described with several variations in his numerous papers on the subject.[2]

Figure 1 illustrates the typical distribution of herpes zoster involving the geniculate zone. In this case the cutaneous eruption was not associated with facial paralysis (type 1 of geniculate herpes, according to Hunt's classification).

PRIMARY SITE OF THE HERPETIC INFECTION

Symptoms indicating involvement of the inner ear were the first neurologic manifestations noticed in the case at issue. This fact indi-

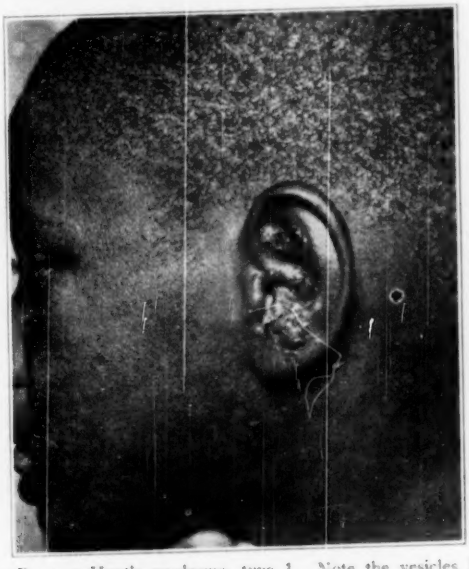

Fig. 1.—Ramsay Hunt's syndrome, type 1. Note the vesicles located over the "geniculate zone," including the concha, external meatus, root of the helix, crura of the antihelix, intercrural fossa, antitragus and external surface of the lobule at the upper part.

cates primary involvement of the acoustic ganglions, as was noted by Johnson and Zonderman. It is a rare observation, recorded in Hunt's series only five times.[21]

The cerebrospinal lymphocytosis, with an unusually high count in the case, is an important diagnostic factor. In instances in which herpetic vestibulocochlear ganglionitis is not associated with facial paralysis and/or herpetic eruption, it may be particularly helpful for differential diagnosis of this condition versus labyrinthine hydrops and/or acoustic neuroma.

ANATOMIC FACTORS INVOLVED IN THE PATH OF EXTENSION TO THE SEVENTH NERVE

Johnson and Zonderman assumed that the process "in all probability involved the facial nerve via the communication between the eighth and seventh in the inner auditory meatus." A brief discussion of some anatomic peculiarities concerning this anastomosis seems to be indicated. It may provide an explanation for the suprageniculate syndrome of facial nerve lesion, which caused the authors some difficulty in its classification. According to Cunningham,[4] this anastomosis is provided by two nerve filaments (fig. 2). One of them crosses via the pars intermedia of Wrisberg and joins the seventh nerve in the internal auditory meatus. The other, crossing directly, joins the seventh nerve

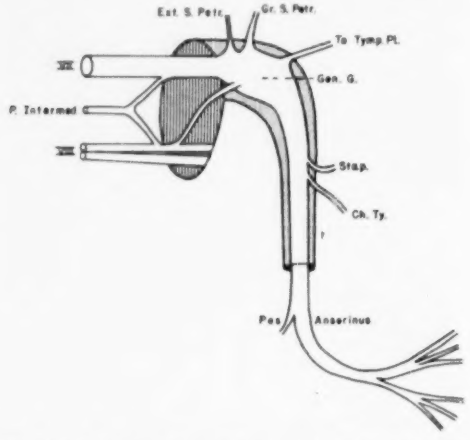

Fig. 2.—The eighth to seventh nerve anastomosis is provided by two filaments, one passing via the pars intermedia in the inner auditory meatus and the other crossing directly to the geniculate ganglion in the fallopian aqueduct.

in the aqueduct of Fallopius at the site of the geniculate ganglion. For the purpose of this analysis, I shall call the former the "suprageniculate" and the latter the "transgeniculate" anastomosis.

It should be clearly understood that an inflammatory process extending from the eighth to the seventh nerve across the "suprageniculate" bridge will necessarily attack exclusively those nerve fibers which constitute the seventh nerve at this location, namely, the motor and the secretory portion representing the two efferent nerve structures of the facial nerve and the sensory root which, originating in the geniculate ganglion, represents the only afferent nerve structure at this site of the

4. Cunningham, D. J.: Text-Book of Anatomy, ed. 2, New York, W. Wood & Co., 1906, p. 687.

seventh nerve. There are no gustatory fibers to be injured at this location, since these fibers leave the course of the facial nerve at the site of the geniculate ganglion.

On the other hand, an inflammatory process when passing from the eighth to the seventh nerve across the "transgeniculate" bridge will necessarily attack those nerve fibers which constitute the seventh nerve at the site of the geniculate ganglion, namely, both pairs of afferent and efferent nerve structures.

Assuming in the case of Johnson and Zonderman extension of the herpetic inflammation via the "supragenicular" bridge, the syndrome of the facial nerve lesion manifested in their case appears to be satisfactorily explained. The mild herpetic eruption indicates involvement of the sensory root; the muscle paralysis indicates involvement of the motor fibers, and the ipsilateral lack of lacrimation and dryness of the tongue (as I, too, like to assume) indicate involvement of the secretory fibers. Presence of taste sensation fits into this assumption.

As an interesting counterpart to the present case, the report of a case presented by Hammerschlag [5] comes to mind. This author recorded signs which on the basis of my analysis are suggestive of a possible extension of the herpetic inflammation across the "transgeniculate" bridge. Hammerschlag described a man 32 years old, who became sick five days after exposure to cold. The first symptoms were chills, nausea and vomiting, vertigo, headache, pain in the right ear with roaring sounds and diminished hearing and loss of equilibrium. Two days later, facial paralysis on the right and ipsilateral lack of lacrimation were noticed. Simultaneously herpes vesicles appeared at the external ear. There was a mild swelling of the gums on the same side. Taste was lost over the anterior two thirds of the tongue.

In this case absence of lacrimation plus absence of taste at the chorda tympani area of the tongue places the site of the facial nerve lesion at the geniculate ganglion. Hammerschlag's case demonstrated that a herpetic inflammation involving primarily the acoustic ganglions can be followed by facial paralysis such as manifesting the "transgeniculate" syndrome of a seventh nerve lesion—unlike the case of Johnson and Zonderman. In Hammerschlag's case extension of the inflammatory process across the "transgeniculate" bridge is offered as a possible explanation.

Of course, such a concept is merely hypothetic. However, a theory when built on the firm ground of anatomic facts appears well founded, even though it cannot be guaranteed.

CLASSIFICATION

The preceding comments may aid in the appropriate classification of the case of Johnson and Zonderman and at the same time resolve the uncertainty faced by them. To me it appears that their first idea which

5. Hammerschlag, V.: Beitrag zur Casuistik der multiplen Hirnnervenerkrankungen, Arch. f. Ohrenh. **14**:1, 1898.

"places this case in the rare group 4 of Hunt's classification of 'geniculate zoster'" is correct. There is no need to drop it because of the topognostic analysis indicating the site of the facial nerve lesion as supragenicular. The preceding comments make it evident that this location is entirely compatible with findings recorded in the case by Johnson and Zonderman. Moreover, this case reveals the interesting fact that mild herpetic eruptions in areas supplied by the sensory portion of the seventh nerve may occur without presenting topognostic signs characteristic of a lesion of the facial nerve at the geniculate ganglion but presenting signs characteristic of a supragenicular lesion.

Considering the site(s) of the herpetic eruptions the case of Johnson and Zonderman is to be classified as an example of Hunt's syndrome type 4.

Considering the ganglions primarily involved by the herpetic infection, the case should be regarded as an instance of herpetic ganglioneuritis acoustica, the inflammation very likely extending onto the facial nerve across the supragenicular branch of the eighth–seventh nerve anastomosis.

CRITERIA FOR RESTRICTION OF THE TERM RAMSAY HUNT'S SYNDROME

Johnson and Zonderman finally recommended restriction of the term "Ramsay Hunt's syndrome" or "geniculate zoster." The identical idea was suggested for the first time in my article as the result of studies on the subject. However, the criteria for determination as suggested in my previous paper and those suggested recently by Johnson and Zonderman are different. Since the pertinent passage in my previous article is not mentioned by Johnson and Zonderman, correlation of the two different viewpoints may be facilitated by direct quotation from both articles.

Tschiassny

It is suggested to restrict the term "Ramsay Hunt's syndrome" exclusively to instances presenting herpes zoster in the "geniculate zone" of the external ear.

Johnson and Zonderman

It is emphasized that the term "Hunt's syndrome" or "geniculate zoster" should be restricted to cases showing definite involvement of the geniculate ganglion as evidenced by histopathologic study or topognostic analysis.

In my concept, the site of the herpetic eruptions is regarded as exclusively significant in classification of the clinical entity at issue. In the concept of Johnson and Zonderman, proof of involvement of the geniculate ganglion is required. Evidence in this concern may be obtained in either one of two ways: (*a*) by histopathologic study or (*b*) by topognostic analysis. It is necessary to determine whether requirements such as postulated by the authors are justified.

Evidence from Histopathologic Study.—The importance of histopathologic examination of the geniculate ganglion in cases of herpes zoster oticus cannot be overemphasized. It certainly represents the

most reliable way of resolving some of the problems regarding the subject. However, in the individual case, utilization of findings obtained by microscopic studies is restricted to diagnosis *in mortuo*. Moreover, autopsies are rarely obtained in cases of herpes zoster, since it is per se not a deadly disease. Investigation in this concern depends on fatality intervening at the time when the zoster infection is present. With regard to herpes zoster at the auricle, there are so far only 3 autopsies recorded in the literature. Unfortunately, in the first case [3e] (Hunt) the part of the facial nerve at the site of the "intumescentia ganglionaris" was lost. In the second case [6] (Maybaum and Druss) the simultaneous presence of a suppurative process involving the middle ear and the inner ear structures opened the door for equivocal interpretations of the histopathologic changes noticed in the geniculate ganglion; no objections can be marshaled against the third case, reported by Denny-Brown, Adams and Fitzgerald.[7] It was a case of herpes occipitocollaris. Pathologic changes were demonstrated in the upper cervical ganglions. The geniculate ganglion appeared to be normal.

From these reports it becomes evident that histopathologic changes in the geniculate ganglion in association with Ramsay Hunt's syndrome have not so far been shown unequivocally. For this reason it appears not justifiable to postulate demonstration of such changes as a requirement for the diagnosis of this clinical entity.

Evidence from Topognostic Analysis.—The idea of restricting the terms to instances in which the involvement of the geniculate ganglion appears to be proved by topognostic analysis seems to be based on my original studies methodically performed in a larger series of cases and referred to by Johnson and Zonderman. Unfortunately, my findings and the conclusions obtained therefrom seem to have been misinterpreted in their article. This conjecture is particularly based on figures and statements quoted by them which, however, do not conform to those presented by me.

Johnson and Zonderman asserted "Reviewing 22 cases of herpes zoster with facial paralysis (Hunt's syndrome) in the literature with regard to the topognostic signs, Tschiassny found that in only 7 of them could the lesion be placed in the geniculate ganglion, in contrast to the tenets of Ramsay Hunt." This statement does not conform with figures presented in table 2 of the article referred to by them.

This table shows that in 22 cases in the literature, 7 authors reported the rash in the geniculate zone. In only 5 of those 7 could the lesion of the facial nerve be placed at the geniculate ganglion, not in all 7 of them as erroneously claimed by Johnson and Zonderman.

6. Maybaum, J. L., and Druss, J. G.: Geniculate Ganglionitis (Hunt's Syndrome), Arch. Otolaryng. **19**:574 (May) 1934.

7. Denny-Brown, D.; Adams, R. D., and Fitzgerald, P.: Pathologic Features of Herpes Zoster: A Note on "Geniculate Herpes," Arch. Neurol. & Psychiat. **51**:216 (March) 1944.

In 2 cases of herpes at the geniculate zone, interference with lacrimation was not present, and therefore on the basis of this method of topognosis the site of the facial nerve lesion was to be placed below the geniculate ganglion. I am not in a position to offer a reliable explanation for the absence of lacrimation in those 2 cases. It seems, however, that the "dry eye" is sometimes only a transient phenomenon which may disappear after a few weeks (maybe even sooner), as was observed in the cases reported by me and by Johnson and Zonderman.

In this connection I wish to call attention to the importance of checking for the presence of the stapedius reflex as a means of differentiating a nuclear lesion of the seventh nerve from an infrachordal lesion (below the branching of the chorda tympani). In either one of these two locations, taste and lacrimation are present. The stapedius reflex, however, is absent when the site of the lesion is in the motor nucleus, while it is present when the lesion is located below the branching of the stapedial nerve and, of course, below the chorda tympani. Absence of the stapedius reflex is manifested by "phonophia"—a painful sensation which is felt when intensities of 60 decibels above threshold are applied, particularly at the frequencies of 512 and 1024 cycles per second.

TABLE 2*

Site of Herpetic Rash	Number of Cases	Site of Facial Nerve Lesion			
		Infrachordal	Suprachordal	Transgeniculate	Suprageniculate
Occipitocollar	10	5	5	..	..
Facial	5	2	2	..	..
Geniculate	7	..	2	5	..

* From Tschiassny.[3]

By these topognostic studies evidence was obtained that the facial nerve lesion is located at the site of the geniculate ganglion in the overwhelming majority of instances of geniculate herpes. In no case of facial or occipitocollar herpes were there signs indicative at this site of a facial nerve lesion.

On the basis of this statistical study, I concluded that in the groups presenting the rash in areas supplied by gasserian or by cervical nerve fibers the geniculate ganglion is not involved at all—contrary to Ramsay Hunt's theory—whereas in the group presenting the rash at the external ear in the area supplied by the sensory portion of the seventh nerve, according to Hunt and designated by him as "the geniculate zone," "the geniculate ganglion seems to be involved, conforming with Ramsay Hunt's theory." By this reasoning it seemed to me justifiable "to restrict the term 'Ramsay Hunt's syndrome' exclusively to instances presenting herpes zoster in the 'geniculate zone' of the external ear."

Topognostic studies as reported previously can be regarded only as illustrative of the problem in general. Performed on a larger scale, these studies provide information in three concerns: 1. Occipitocollar herpes. In no case could the facial nerve lesion be located at the site

of the geniculate ganglion. This fact reflects clinically the histopathologic findings of Denny-Brown and co-workers, who in their case demonstrated normal conditions at this ganglion. It also confirms their conclusion that evidence for geniculate ganglionitis in Hunt's opinion being responsible for facial paralysis in herpes zoster of the head and neck is invalid. 2. Gasserian herpes. On the basis of identical clinical findings concerning the site of the facial nerve lesion in cases of this type of herpes zoster, identical conclusions appear to be justified. 3. Geniculate herpes. In the overwhelming majority of the cases included in this group the facial nerve lesion could be located at the geniculate ganglion. Accordingly, Ramsay Hunt's concept that geniculate ganglionitis is responsible for the facial nerve lesion appears clinically supported in this group of herpes zoster.

However, topognosis cannot be regarded as a method reliable for classification when applied to the individual case. This statement is based on three arguments:

1. The site of the facial nerve lesion in 8 cases (7 in the literature and 1 of my own) of herpes zoster at the geniculate zone could not be demonstrated at the geniculate ganglion in 2 instances.

2. Involvement of the geniculate ganglion by topognostic analysis of a facial nerve lesion depends, of course, on the presence of such a lesion. It, therefore, cannot possibly be demonstrated in instances representing the type I of Ramsay Hunt's syndrone. Accordingly, acceptance of such a requirement would eliminate from the group determined as "geniculate zoster" cases such as the one pictured in figure 1, and yet this case represents almost a textbook picture of this condition.

3. The case reported by Johnson and Zonderman provides new and important information. It demonstrates that herpes eruption, even in both the auricular and the glossopalatine area of the "geniculate" distribution, may occur with topognostic signs indicating the facial nerve lesion not at the site of but above the geniculate ganglion. Accordingly, their case represents an additional and a new type of exception to the rule previously mentioned. It probably may even be regarded as another blow to Hunt's theory—presupposing, of course, my analysis to be correct.

It might be justifiable to classify Ramsay Hunt's syndrome according to the site of the herpetic eruption, disregarding the neurologic signs which sometimes may be absent.

CONCLUSIONS

Restriction of the term "Ramsay Hunt's syndrome" or "geniculate herpes" to cases showing involvement of the geniculate ganglion as evidenced by histopathologic studies or topognostic analysis does not appear to be tenable. These terms should rather be restricted to instances presenting herpes zoster at the geniculate zone of the external ear, as previously suggested by me. Of course, the rare occurrence of oropalatine involvement should also be considered.

The interesting case reported by Johnson and Zonderman represents an important contribution to the subject. The dissenting views discussed in this paper do not deduct from the value of their report.

I appreciate the credit paid to me by the authors, particularly with regard to the method concerning topognosis of facial nerve lesions. However, I wish to emphasize that my contribution in this regard represents merely a simplification of Wilhelm Erb's[8] pioneer work on the subject.

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8. Erb, W. H., in von Ziemssen, H.: Cyclopedia of the Practice of Medicine, translated by R. H. Fitz, C. P. Putnam, and others, New York, W. Wood & Co., 1876, vol. 11, p. 490.

DACRYOCYSTORHINOSTOMY

Report of One Hundred and Fifty Cases of Dacryocystitis Occurring from 1938 to 1947

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BROOKLYN

HISTORICAL REVIEW

ALL HISTORICAL cultures had given attention to abnormal conditions of the eye, to a greater or lesser degree. Increasing research in the field of archeology will undoubtedly show this to be true, and so it is not surprising to find that attempts to relieve the discomfort attendant on suppuration of the tear sac and the accompanying tearing date back to remote antiquity.

Of the many historical cultures, this paper is concerned only with the Greek, Arabian and Western. They, in turn, originated or borrowed ideas and technics from their neighbors or predecessors. The mark of a culture is not the fact that it borrowed a technic but what it did with it and how it developed the technic until it reached its ultimate perfection.

The Greek and Arabian cultures attacked the problem externally, because of their lack of intimate knowledge of the inner topography of the nose. The idea of anastomosis must be accredited to Greek thought; it was in turn taken over and elaborated by Arabian medicine and carried to its present refinement by the technical improvements provided by Western civilization.

The surgical approach to the problem has, in modern times, been directed to intranasal procedures. Inasmuch as I have always favored the external route, I will confine myself to historical data concerned with the external operation.

Greek Culture.—In the period from 30 to 50 A.D. Aurelius Cornelius Celsus in his "De medicina," a section of a greater work, "De artibus," probably established the principle of fistulization into the nose by means of the cautery. Six centuries had elapsed between this and the earlier writings of Hippocrates. One thousand years earlier Egyptian medicine had very likely reached its peak. Claudius Galen (130-200 A.D.) quoted Arrhigens (97-117 A.D.), a contemporary of Trojan, as follows: "When the eye is sick in the middle, one cuts the median ocular angle, spreads the wound, then bores with a small borer many small

From the Brooklyn Eye and Ear Hospital (service of Dr. F. L. Tucker).
Read before the American College of Surgeons at the Brooklyn Eye and Ear Hospital, October 1947.

holes until the bony wall is broken; this cures the sickness." An alternative method is also mentioned. "Lay the bone free and then press a glowing iron against the bone. This causes the bone to fall away and the sickness is cured." Still another method is described, in which a funnel is placed in contact with the tissues and cautery made with molten lead. In the year 700 A.D. a Greek doctor, Paul of Aegina, described a similar operation, probably copied from Galen: "The skin near the nose is cut and the bone is perforated."

Arabian Culture.—The greatest period of Arabian cultural development came during the period from 600 to 800 A.D. However, its technical and scientific advances continued on for several centuries. During this period of greatest Arabian influence, about 1000 A.D., 'Ali ibn 'Isa of Bagdad wrote a textbook on ophthalmology, in which for the first time a triangular instrument was described for trephining the nasal bone. Blood flowing from the nose was the test of a successful operation. 'Ammar of Cairo treated fistulas of the sac, when the neighboring bone was infected, by perforating the nasal bone with a glowing hot cautery. The test for completion of a successful operation was smoke emanating from the nostrils.

Western Culture.—From the year 1000 A.D. through the Middle Ages nothing new could be added to the story. The hot cautery remained the method of choice. It was used and described during this period by Guy de Chauliac, Ambroise Paré and Hieronymus Fabricius. For seven centuries after the advent of Arabian medicine the underlying principle had not been changed. The centuries produced no craftsmen who modified or improved the procedure.

The eighteenth century saw a rebirth of ophthalmology, and with it some serious attention was given the problems concerned with the cure of chronic dacryocystitis. In 1724 Woolhouse, an Englishman who worked in Paris, described an operation in which he discarded the cautery and trephined the nasal bone with a golden cannula, at the same time draining the sac into the nose. His pupil Platner continued to practice the procedure and tried to popularize it. Berlin, von Graefe, Kuhnt, Czernak, Axenfeld and Mueller also tried to popularize this procedure, but it soon lost its popularity and even they themselves reverted to extirpation of the sac. This method has enjoyed popularity to the present day. Even though a vast majority of the patients were cured of suppuration, all of them continued to have epiphora. This annoying situation left them dissatisfied and unhappy.

In 1904 Toti,[1] of Florence, revived the old trephine operation of Woolhouse and Platner. He made a bony window, with care not to

1. Mosher, H. P.: Combined Intranasal and Extranasal Operation of the Lacrimal Sac (Mosher-Toti), Ann. Otol., Rhin. & Laryng. **32**:1-14 (March) 1923.

injure the nasal mucosa. He removed the inner wall of the sac, and the nasal mucosa opposite was also excised. No sutures were used, but contact was made with a tampon. The ethmoid cells were not stressed. He stated that results were good in 50 per cent of the cases. Strazza, Elschnig and Lagrange confirmed Toti's results and upheld the value of the operation. It was again advocated in 1926. In 1914 Kuhnt [2] suggested sutures in making the anastomosis between the nasal mucosa and the sac.

In 1920 and 1921 Dupuy-Dutemps and Bourget reported their operation of dacryocystorhinostomy with the use of accurately placed sutures. However, they did not stress the ethmoid cells as a problem and did not focus the attention of the profession on them as a factor in the successful performance of the operation. It was a definite advance on the Toti method. They reported successful results in about 95 per cent of cases. Dupuy-Dutemps continued this work with intermittent reports to the medical profession, and on May 1933 he was able to report 1,000 cases of dacryocystorhinostomy, with a 94.8 per cent rate of cure for both suppuration and epiphora. These statistics did not influence the ophthalmologic profession.[3]

In March 1923, after a trip abroad, Mosher [1] returned with the Toti-Mosher operation, the Toti operation being performed through an external incision and the curettage of the anterior ethmoid cells, with the removal of the anterior end of the middle turbinate body intranasally. He stressed the importance of the ethmoid cells as a factor in the success of the operation. According to Rychener [4] of Memphis, Tenn., good results had been reported in from 40 to 100 per cent of cases. Averbakh and Ivanova [5] reported 1,200 observed cases with successful results in 96 per cent (1935).

Space does not permit me to enumerate all the men [6] in the last twenty-five years who performed this operation and who reported successful results in more than 90 per cent of cases, in large and small series. But sufficient evidence has been presented to show that in com-

2. Kuhnt, cited by Hallum, A. V.: Dacryocystorhinostomy: Logical Treatment of Occlusion of Lacrimal Sac, J. M. A. Georgia **32**:186-189 (June) 1943.

3. Dupuy-Dutemps, L.: Observations sur mille dacryostomies plastiques, Ann. d'ocul. **170**:361-384 (May) 1933.

4. Rychener, R. O.: Present Status of Surgery of Lacrimal Sac, Surg., Gynec. & Obst. **68**:414-418 (Feb.) 1939.

5. Averbakh, M., and Ivanova: Douze cents opérations de dacryocystorhinostomie plastique, Ann. d'ocul. **172**:913-936 (Nov.) 1935.

6. Hughes, W. L.; Guy, L. P., and Bogart, D. W.: Dacryocystorhinostomy, Surg., Gynec. & Obst. **73**:375-380 (Sept.) 1941. Chandler, P. A.: Dacryocystorhinostomy, Tr. Am. Ophth. Soc. **34**:240-263, 1936. Yanes, T. R.: Dacryocystorhinostomy: Interesting Points on Operation, Arch. Ophth. **26**:12-20 (July) 1941. Traquair, H. M.: Chronic Dacryocystitis: Causation and Treatment, ibid. **26**:165-180 (Aug.) 1941.

petent hands this operation is preeminently successful. It has a vogue and popularity in the larger institutions but is still a nonacceptable procedure with the rank and file of ophthalmologists. Dacryocystectomies are still being performed in great numbers. Dupuy-Dutemps [3] had the following to say about his operation and the attitude of his colleagues:

> It is astonishing that the majority of ophthalmologists still ignore the great value of this operation and persist in doing a dacryocystectomy in the case of a suppurative sac. Some do not even mention its possibility or indication to the patient. In view of the extraordinary good results that I have obtained, it becomes the duty of every good ophthalmologist to indicate and do this excellent surgical procedure. This state of affairs is all the more astonishing because the medical world has searched for many hundreds of years for an operation that would be a satisfactory cure for this condition. Now that we have it, it is for the most part ignored. Thus the result is that our patients are condemned forever to suffer from tearing and watery eyes.

This statement made by Dupuy-Dutemps in France before the Paris Ophthalmological Society fifteen years ago is true today in the United States. Cystectomies are still done in the majority of cases because they are easier to do, to the great loss and later suffering of the patient. A dacryocystectomy is not a good surgical procedure and should disappear at once from ophthalmologic practice.

OSTEOGENESIS OF THE BONY WINDOW

It is obvious that the success of the operation depends on an adequately large bony window. This is only another way of saying that a bony fistula is made through which the anastomosis between the sac and the nasal mucosa can be formed.

If the area in which the window is to be made consisted of homogeneous lamella of bone of varying thickness it would simplify matters and the window would be made easily with average instruments and in a size required to meet the needs of the particular patient at the time. Most textbooks imply that this is so, and all the earlier operators described their procedures as if there were no further obstacles.

However, this is not so. The important structure hindering the successful creation of the window is the ethmoid cells. They almost never are found in the place ascribed to them in the standard texts. It is a matter of simple investigation, for any one who has access to skulls, to examine the region of the lacrimal bone, the nasal process of the maxilla and its junction with the nasal bone to see that the ethmoid cells can and do extend as far anteriorly as the nasal bones. This is extreme, but nevertheless it does occur in a fair percentage of skulls; in a greater percentage the cells are in contact with the entire lacrimal bone, anterior and posterior to the posterior lacrimal crest. Of course, the vast majority show cells in relation to the posterior half of the

lacrimal bone. One cannot confine oneself to the anterior half of the lacrimal bone in creating the window. The area would be too small, new-formed bone would soon cause pressure on the anastomotic line and failure would undoubtedly ensue.

These facts are realities for me, not only from observation on the cadaver but from encountering extensions of the ethmoid cells to the nasal bones in the course of the series of operations presented here.

Undoubtedly, the earlier operators must have encountered a similar situation and successfully coped with the ethmoid cells when encountered. However, they did not tell us about it in their writings.

Whitnall [7] presented a colored section clearly showing the lacrimal sac in contact with the anterior cells; but he did not state how far forward they extend. I have found them surrounding the neck of the sac almost completely. Certainly, the anterior inferior portion of the lacrimal bone presents a small area about 5/8 inch (1.6 cm.) in diameter free from cells. But for all practical surgical considerations this fact is of no importance and, moreover, should delude no one in contemplating an anastomosis through so small a window. The most that one can do is probably to transplant the neck of the sac into the nose,[8] and osteogenesis would soon defeat that after a short period.

To determine that osteogenesis takes place is a matter of simple palpation, both externally and intranasally, with a probe. I have tried by means of roentgenograms taken postoperatively to determine its course and rate. However, I soon gave this up, because no accurate information could be gathered concerning the identification of the window or its size.

The creation and maintenance of a bony fistula has been a problem in nose and throat surgery for a long time. Small windows in the nasoantral wall for antral drainage are eventually closed by osteogenesis, which repairs the defect. In otologic work, the bony fistula in the fenestration operation closes in a good proportion of the cases through osteogenesis.

The question of how to prevent osteogenesis has not been solved. In working with the lacrimal sac I have been making a large window, so I believe that osteogenesis exhausts itself before the window can close; in addition, I believe that sewing the mucosal flaps over the window edges helps by slowing the rate at which this happens and exhaustion takes place sooner.

In addition, I have practiced careful removal of the ethmoid cells with their mucosal lining. This cannot be done crudely with the average technic. Each cell wall must be broken down and carefully removed so

7. Whitnall, S. E.: The Anatomy of the Human Orbit and Accessory Organs of Vision, ed. 2, New York, Oxford University Press, 1932, pp. 223-224.

8. Stokes, W. H.: Transplantation of Lacrimal Sac in Chronic Dacryocystitis, Arch. Ophth. **22**:193-210 (Aug.) 1939.

that the nasal mucosa is not injured. In this way, I can make the window of adequate size, remove additional foci of osteogenesis and give adequate mobility to the mucosal flaps for accurate suturing.

In more than 100 cases the middle turbinate had not been removed. I think that except in rare cases removal of the anterior end will not be necessary.

PATHOLOGIC FINDINGS

I have no microscopic sections of the pathologic material, because none was removed at the time of operation, with the exception of some small pieces of polyp-like material that were floating in the distended sacs in 2 cases, causing a ball valve action so that pus could not be readily expressed through the canaliculi. In 1 of these cases there was an internal fistula communicating with the nose so that pus drained through the nose when pressure was made on the sac.

The following changes were noted at the time of operation, and they would largely parallel the findings in the sections obtained in dacryocystectomies:

1. Distended sacs with thickened hyperemic walls, containing mucus or mucopus; strictures at the neck in almost all cases
2. Mucosal hypertrophies
3. Polypoid thickenings of the mucosa
4. Shriveled sacs, so distorted that there was hardly any lumen
5. Erosions
6. Semipermeable strictures at the neck and internal punctum
7. The internal punctum originating at times in the dome of a shriveled sac
8. Pericystic infiltration and thickening in the surrounding soft tissues in the recently or recurring acute cases
9. Skin fistulas
10. Internal fistula through an anterior extension of the ethmoid cells
11. Anterior extension of ethmoid cells, normal or diseased, in many cases (as noted in the discussion on the bony window)
12. The anterior end of the middle turbinate body extending to the incision of the mucosa sutures, in my first series (none in my later series)
13. Polyp-like bodies floating free in the mucopus of the sac
14. Trabeculae of the wall of the sac
15. Granulomas, particularly in the region of the internal punctum
16. Slit canaliculi (no contraindication to operator)
17. Only one patent canaliculus, 3 cases (does not impair a good result)

INCIDENCE OF DACRYOCYSTORHINOSTOMY

The high frequency of successful results for the type of operation involving an actual anastomosis of the sac and nasal mucosa was established by the earlier workers in this field in a large series of cases, 2,000,

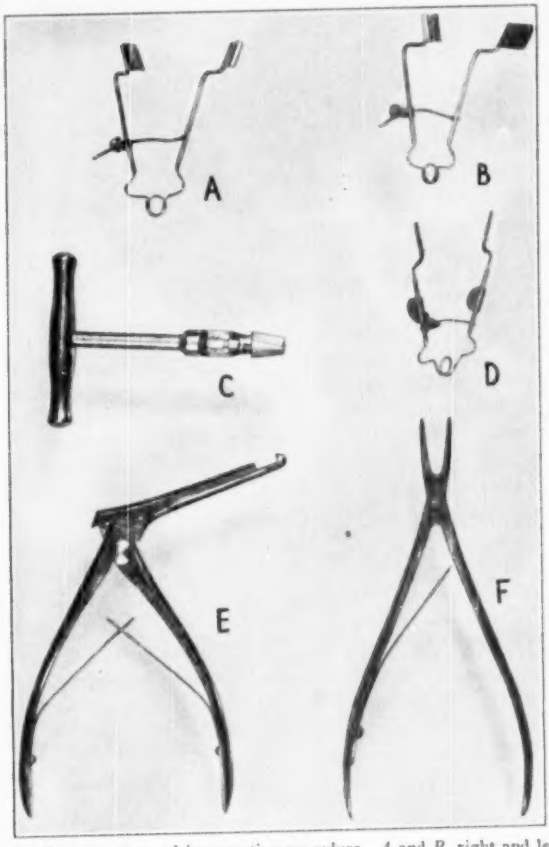

Fig. 1.—Instruments used in operative procedure. *A* and *B*, right and left Welt lacrimal sac retractors; *C*, De Vilbiss trephine; *D*, Welt long tooth lacrimal sac retractor; *E*, Kerrison rongeur, and *F*, Lempert rongeur.

for example. The limited series of cases presented here, 150, gives a similarly large percentage of successful results. These successes have endured over a period of years, and it is reasonable to believe that they will continue. The graph indicates that in the past eight years dacryo-

cystorhinostomy has not been a popular operation with the majority of ophthalmologists. Dacryocystorhinostomies have been performed with appreciable frequency to greater or lesser degree, varying from year to year. It appears that the medical profession still needs to be convinced about the value of dacryocystorhinostomy.

THE OPERATION

The historical evolution of the operations for the relief of chronic dacryocystitis leads to the conclusion that these operations, while theoretically sound, did not in their performance lead to uniformly good results by average operators. Thus these operations both external and internal have enjoyed only a limited popularity, particularly in cities associated with large medical institutions in various parts of the country. They were not uniformly advocated, nor were they performed with regularity. Dacryocystectomy was the operation of choice in spite of its obvious disadvantages, the chief of which was the ensuing epiphora in the majority of cases.

Being mindful of the surgical principle that it is best to operate by direct vision, I naturally favor the external operation. I also favor the operation of Bourget and that of Dupuy-Dutemps, inasmuch as their use of sutures favored accurate apposition of the parts and quicker healing without exuberant granulations forming in the nasal chamber. I also believe that the variations of the anatomy from the normal and associated pathologic processes in the ethmoid labyrinth could best be handled through the external approach.

In this manner I hoped to standardize the technic of Dupuy-Dutemps and of Bourget, so that with one operative procedure all pathologic conditions and anatomic variations could be corrected in the average case, with little or no postoperative treatment. Thus no additional operative procedures were contemplated or done.

The operative technic is esentially copied from the neurosurgeons. It aims at a bloodless field at all times with complete hemostasis. Suction, the Bovie unit and cottonoid* sponges are used. In addition, I use the regular lacrimal sac retractor with short teeth and another with longer teeth to enable me to retract the orbicularis fibers. Two retractors are used, a right and left, designed by me to protect the sac and soft tisues, while I perform the bone work. The other instruments are the usual ones found in any operating room in which plastic and ear, nose and throat procedures are performed.

My procedure is as follows:

Local anesthesia with 1 per cent butethamine hydrochloride solution (monocaine hydrochloride*) and epinephrine hydrochloride are used in all cases except with children, for whom general anesthesia is used.

I use no preliminary medication, as a rule, except for nervous persons. They receive ⅙ grain (11 mg.) of morphine sulfate and 1/150 grain (0.40 mg.) of atropine sulfate, on call.

In addition, 2 per cent tetracaine hydrochloride solution (pontocaine hydrochloride®) is instilled into the conjunctival sac of the eye on the side to be operated on. The nasal chamber on the same side is painted with 2 per cent tetracaine hydrochloride solution or 10 per cent cocaine hydrochloride solution, with epinephrine hydrochloride (1:1,000) added. This solution is left until the nasal mucosa is incised.

The butethamine solution is infiltrated subcutaneously and subperiosteally along the line of incision, at the lacrimal crest and at the dome of the sac. The area is then gently massaged so as not to distort any landmarks.

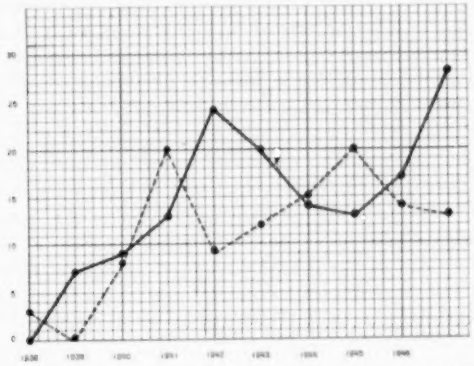

Fig. 2.—Incidence of dacryocystorhinostomy compared with that of dacryocystectomy over a ten year period (1938-1947) at the Brooklyn Eye and Ear Hospital. Unbroken line indicates dacryocystorhinostomy; broken line, dacryocystectomy.

The actual operation does not differ from the ones usually described, but I have tried to combine the best points inherent in both the external and intranasal procedures, stressing certain parts of the technic that I deem important. The operation as performed is divided into several steps that are painstakingly carried out to its completion.

The incision in the skin is a small one, rarely longer than ¾ inch (1.9 cm.). It is the one that I use in performing radical operations on the frontal and ethmoid sinuses. In this instance I curve the lower part laterally so that it terminates at a point over the lacrimal crest. The latter structure coincides with the entrance to the nasolacrimal canal.

The incision starts about 4 mm. above the medial palpebral ligament and is about 5 to 6 mm. lateral to it at its center, the lower part curving

laterally so that it ends at a point about 4 mm. laterally to the inner canthus. The skin only is incised at this point to the deep fascia. The incision is continued to its extremities with a Stevens scissors. The small vessels are clamped and tied so that the field is relatively bloodless. No lateral undermining of the skin is done. The three-pronged lacrimal sac retractor is then inserted and the skin is gently but firmly stretched for about 5/16 inch (0.8 cm.), so that the underlying fascia shows dull and dry. Running below the fascia is noted the external angular vein and its smaller superior branches on or amongst the muscle fibers. Time is taken to tie this vessel or to push it gently outside the field so that the field continues bloodless. The coagulating current has been suggested for this maneuver, but I have not used it.

The orbicularis muscle fibers are then separated along the line of the incision by blunt dissection, either by inserting and separating the blades of a Stevens scissors or a mosquito clamp. This separation is continued down to the periosteum until that structure shows up white and dry in the depth of the wound. Pressure and suction applied against wet cottonoid® sponges will help facilitate this procedure without injury to the soft parts.

Time is again taken to dry the field, using sutures on small curved needles if necessary to obliterate bleeding vessels. The lacrimal sac retractor is now removed and again reinserted so that the teeth engage both the cutaneous and the muscle tissues, and the wound is again firmly dilated to about 5/16 inch.

The periosteum is now incised throughout the length of the incision. The lateral edge is reflected from the nasal process of the maxilla, and as the separation deepens the lacrimal sac is carried along with it. The separation is continued by pressure on cottonoid® sponges so that anterior and posterior lacrimal crests and the nasolacrimal canal are visualized for at least 1/4 inch (0.6 cm.).

The retractor is now removed and another one with a large metal strip inserted to protect the sac in preparation for the creation of the bony window. Sometimes the separation of the periosteum is continued along the infraorbital margin for 1/4 to 3/8 inch (0.6 to 1.0 cm.). Thus the sac can be mobilized well away from the bone, mainly at the level of the anterior lacrimal crest and below it, where the removal of the bone is now necessary to insure a good result.

With the metal retractor well placed so that the wound is widely separated, a trephine opening 5/16 inch in diameter is made, with its center at the posterior border of the nasal process of the maxilla. The standard De Vilbiss trephine is used. The bone disk that is cut is removed by prying it out of the surrounding bone with a suitable elevator. Surprisingly little bleeding occurs with this maneuver.

The trephine opening is the first step in the creation of the bony window. I do not follow any exact measurements but will state that the window should be one and one-half times the area of the sac. The greatest amount of bone is removed in the region of the lower part of the lacrimal fossa. The inner wall of the nasolacrimal canal, formed by the union of the lacrimal process of the inferior turbinate bone, the nasal process of the maxilla and the anterior and the posterior crest of the lacrimal bone, is removed by suitable rongeurs, enabling the operator to visualize the membranous canal. After this step is done the sac will, in most instances, shift medially and will practically be in apposition with the nasal mucous membrane. The Hajek bone forceps, the Citelli bone forceps and the Jansen mastoid bone forceps are used in the removal of the bone in the making of the bony window.

In a certain percentage of cases, on removal of the disk of bone created by the trephine, the ethmoid cells, normal or diseased, are found immediately adjacent to it, and as the bony defect is enlarged these cells must be removed with suitable instruments for ethmoidal surgery. In this instance I would advise their removal by the gentle use of the curet until one can visualize the mucous membrane lining the outer wall of the nose. Bleeding at this stage can be quite profuse and is controlled by means of suction on pledgets of cottonoid* soaked in epinephrine. The spicules of bone are cleared from the wound, and it is noticed that the sac and mucous membrane meet very easily without any tension, quite an advantage when placing the sutures. The retractor is removed and one is now ready for the next step in the procedure.

With a Bowman sound in the lower canaliculus causing pressure so that the inner wall of the sac is made to bulge into the wound, the inner wall of the sac is slit with a scalpel and Stevens scissors from its dome down through the neck and into the canal. The pathologic process is noted and the patency of the remainder of the canal tested with a sound. The nasal packing is now removed and a lead plate inserted into the nose, and the nasal mucosa is incised over it, either in a straight line opposite the incision in the sac or in an H or Y shaped incision, as conditions present indicate. The lead plate is now removed and the anterior end of the middle turbinate bone may be removed with the nasal snare if it projects over the line of incision.

The posterior lip of the nasal mucosa and the posterior lip of the sac are sutured with three interrupted Davis and Geck chromic sutures with atraumatic needles. Three similar sutures are used to suture the anterior lip of the sac wall to the anterior lip of the nasal mucosa. The wound is now inspected for bleeding and sulfadiazine powder dusted over the suture line. The orbicularis muscle is closed with three interrupted plain 0000 surgical gut sutures, and the skin is closed with three or four dermal plastic sutures. A small dry dressing is then applied.

Postoperative care entails the use of suitable sedatives for pain and continuous cold boric acid compresses for the swelling. Nitromersol N. F. (metaphen®) solution (1:2,500) is instilled into the eye three times a day and 2 per cent phenylephrine hydrochloride solution (neosynephrine hydrochloride®) is instilled into the nostril. The patient is allowed up the next day and the skin sutures are removed on the third day. From then on there is no further postoperative treatment until the third week. Then, if the patency of the canaliculus is questionable, due to epiphora, gentle dilation with a sound followed by irrigation with isotonic sodium chloride solution is done.

STATISTICAL SUMMARY

One hundred and fifty cases of chronic suppurative dacryocystitis are presented: In 149 cases the suppuration was cured; in 8 cases the epiphora was not relieved, and in 1 case suppuration recurred after one and one-half years. In this case I used brain clips to suture the posterior lip of the anastomosis. In 140 cases the suppuration and epiphora were cured. Two cases in this series were revisions of work done elsewhere, and the epiphora was not cured, but the suppuration was. Omitting 3 cases leaves a total of 147 cases with failure in 7. The percentage of failure was 4.6. The rate of cure was 95.4 per cent. Patients were observed for ten years and remained well. Postoperative treatment in the 7 cases in which the epiphora was not relieved proved unsuccessful.

The only permanent complication was 1 case of epicanthus, with an otherwise excellent result. The immediate postoperative complications were as follows:

10 cases of mild wound infection which was easily controlled, and
2 cases of intracanalicular conjunctivitis, also easily controlled
2 cases of postoperative nasal hemorrage
4 cases of acute exacerbation of chronic conjunctivitis
10 cases of erosions of the cornea
2 cases of erosions of the cornea, occurring ten days after operation.

The ages of the patients ranged as follows: The youngest patients were from 15 to 18 months of age; 3 patients were 3, 8 and 12 years, respectively, and the oldest patient was 81 years.

SUMMARY AND CONCLUSIONS

One hundred and fifty cases are presented.

Obstacles to the successful performance of the operation are indicated and stressed.

A technic copied from neurosurgical procedures is outlined.

The cause of the failures is indicated as a combination of osteogenesis and infection, coupled with faulty technic. The exact cause cannot always be ascertained.

The high incidence of success in 95.4 per cent of these cases compares with that of the series of Dupuy-Dutemps with success in 94.8 per cent of cases.

There is no contraindication to the operation, and I urge its performance in the presence of ophthalmic infections, such as corneal ulcers, erosion or abscess.

I am cognizant of all the other operations proposed for the relief of suppuration of the lacrimal sac and find nothing in them to compare with the operations proposed by Dupuy-Dutemps and Bourget, which satisfy all indications.

It is hoped that this presentation will help do away entirely with excisional operations practiced by physicians.

769 St. Marks Avenue.

NEW NEUROSURGICAL APPROACH FOR TREATMENT OF OTOSCLEROSIS

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VALENCIA, SPAIN

AS A NEUROSURGEON, I had the pleasure of assisting in the first 5 fenestration operations performed in Valencia, Spain. Having no otologic training and appreciating the difficulties of the endaural approach to the labyrinth, I made investigations which resulted in a new neurosurgical approach to the middle ear and labyrinth. This approach was devised not only for the fenestration operation but also for all procedures in which access to the middle ear and labyrinth is required—labyrinthectomy and others. After a number of experimental attempts on cadavers, the procedure was used on a patient who had otosclerosis but who would be considered a very poor risk for the fenestration operation.

TECHNIC

Scophedal (a preparation containing 0.5 mg. of scopolamine hydrobromide, 10 mg. of dihydrohydroxycodeinone hydrochloride and 25 mg. of racephedrine hydrochloride per cubic centimeter), 0.5 cc., is given intramuscularly a half-hour before the operation and the patient's right temporoparietal region shaved and cleaned. Procaine and epinephrine hydrochloride are used for local anesthesia. An incision is made in the skin and temporal muscle up to the bone. The incision is perpendicular to the zygomatic arch and extends from the upper part of the helix upward for about 8 cm. (fig. 1). The temporal muscle is elevated and the bone bared. A craniotomy as close as possible to the floor of the middle fossa is performed with a gouge of about 3 or 4 cm.

The dura is elevated until the eminentia arcuata becomes clearly visible; this is about 3 cm. from the cranial wall. The tegmen tympani now becomes visible just lateral to the eminence. The porosity of the bony tegmen is characteristic at this point.

With use of an electric burr the tegmen is thinned down and perforated. This perforation is then enlarged with a curet until the head of the malleus and the lateral semicircular canal become visible. The final hole should be about 10 to 15 mm. in diameter. At this point, a system of continuous irrigation and suction is instituted and maintained until the conclusion of the operation. The head of the malleus is disarticulated from the incus. This can be done with great ease through this approach. Sometimes it may not even be necessary to remove the incus; however, to prevent ossicular stiffening, it is recommended that the incus be removed. From now on the creation of the fenestra with use of an electrical

This manuscript was revised by Joseph Sataloff, M.D., of Philadelphia.
Presented at the first meeting of the Luso-Spanish Society of Neurosurgeons, Barcelona, Spain, April 3, 1948.

burr follows the technic developed by Lempert; the window is made in the anterior portion of the lateral semicircular canal (fig. 2).

When the fenestration is completed, a piece of temporal bone is placed over the perforation in the tegmen tympani in order to prevent herniation of the dura into the middle ear. The fenestra nov-ovalis lies about 4 mm. from the floor of the middle cranial fossa. The muscle is then sutured, and the facia and skin are closed.

The advantages of this neurosurgical approach should be considered. The anesthesia is extremely simple, consisting almost purely of local

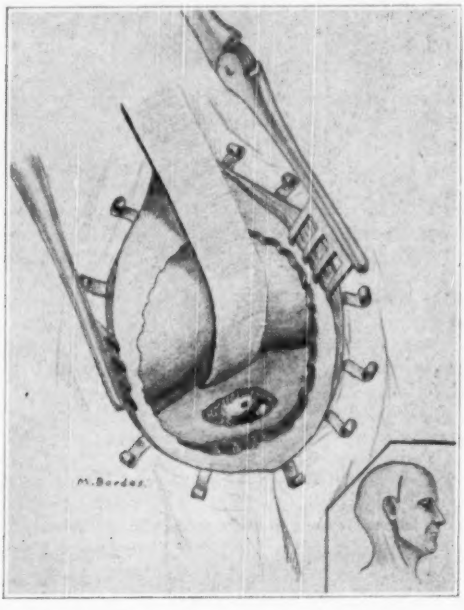

Fig. 1.—The large illustration shows the completed fenestra; the inset, the incision.

infiltration, and there is no discomfort to the patient during the operation. There is a tremendous reduction in the time required for the entire procedure, since very little boring has to be done. This procedure is similar to the decompression of Cushing and the procedure for trigeminal section of Frazier, and consequently it is well known to all neurosurgeons.

The point selected for the opening of the tegmen tympani is not arbitrary, for it is easily identified because of its relation to the arcuate

eminence and the porosity of the bone, which are constant. Furthermore, as has been indicated, there is no need to remove the head of the incus. The visualization of the semicircular canal is almost perfect, since the canal is so close to the floor of the cranial fossa. There is practically no loss of blood. The creation of the fenestra is carried out as described by Lempert and the opening burnished with a gold burr. In our first case I did not leave a flap over the fenestra, which was left exposed in the middle ear. I can be criticized for not placing a flap because of the possibility of infection and initiation of a serious labyrinthitis, as studied by Passow and later Bárány. However, with

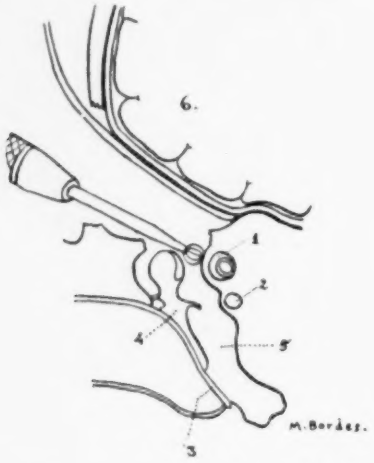

Fig. 2.—Schematic representation of the production of the fenestra. *1*, External semicircular canal; *2*, facial nerve; *3*, tympanic membrane; *4*, malleus; *5*, middle ear; *6*, brain.

my technic the chances of infection are almost nil, and there is no fear of labyrinthitis.

In analyzing some statistical data available at this time I have found that Lempert had 99 closures of the fenestra in 300 cases when the fistula was produced in the loop of the lateral semicircular canal. However, when the fenestra was made in the cupula of the vestibule, in 700 cases there were only 55 closures. Shambaugh failed in 37 per cent of his cases when making the fenestra in the loop but in only 11 per cent of cases when the cupula of the vestibule was used, despite the fact that skin flaps were used in the majority of cases. From this it can be deduced that the flap is not the major factor in causing subsequent

closures of the fenestra, but it is rather the size of the window, since the cupula allows more room for making it larger. The technical difficulties in making the skin flap and the harmful effects of bone particles must also be considered as detriments in the fenestration operation. I therefore consider that my procedure has no reason to produce worse results than the procedure utilizing the skin flap. Another advantage of my procedure is the obviation of any involvement of the facial nerve, since the lateral canal protects the nerve completely. Furthermore, the incision is closed primarily and the integrity of the middle ear retained. In addition, another advantage over Lempert's technic is that the operation can be done when there are adhesions in the middle ear or distortions of the drum membrane. There is also no need to wait for epithelization, and there is no bothersome drainage postoperatively. There is the cosmetic advantage that the scar disappears when the hair grows back. The time of the operation is considerably reduced.

REPORT OF A CASE

In March 1948 I examined a 34 year old woman who complained of an insidious progressive loss of hearing in both ears for the past ten years, worse in the left ear. Recently she had begun to notice some dizziness. There is no familial history of deafness. The patient was examined by Professor Bartual at Valencia and the following notes recorded: The drum membrane was thin and atrophic. The response to the Rinne test was negative on both sides. In the Weber test perception was lateralized to the right. The Schwabach test showed a hearing time of twelve seconds on the right and twenty-five seconds on the left. Conversational voice was recorded at 25 cm. on the left and 0 cm. on the right. The response to the Gellé test was negative on the right and positive on the left. The preoperative audiogram is reproduced in figure 4, curves *A* and *C*. The sharp drop at the 2048 frequency and above should be carefully noted. (It can be seen that this case did not represent an ideal risk for a fenestration operation; however, it was selected to show the facility with which my technic can be performed. I was not interested particularly in the hearing results.)

The right ear was operated on, since the best results could be expected from this side. The hearing seemed to improve when the fistula was produced. The patient experienced dizziness and nystagmus at the same time. After the operation, she was turned on her right side to prevent flow of blood into the labyrinth. The postoperative course was uneventful. Dizziness and nystagmus disappeared on the second day. The temperature (fig. 3) remained normal though the patient was not given antibiotics. She was discharged on the third day. On the eighth day Professor Bartual again recorded his observations. There was slight injection of Schrapnell's membrane, and no exudate was present in the ear. Hearing in the Weber test was lateralized to the right, the response to the Rinne test was negative on the right; the Schwabach test showed a hearing time of twelve seconds on the right. Normal conversation on the right was now heard up to 15 cm. The postoperative audiogram is shown in curves *B* and *D* of figure 4. It can be seen that the greatest improvement was at the 1024 frequency. In the curve for bone

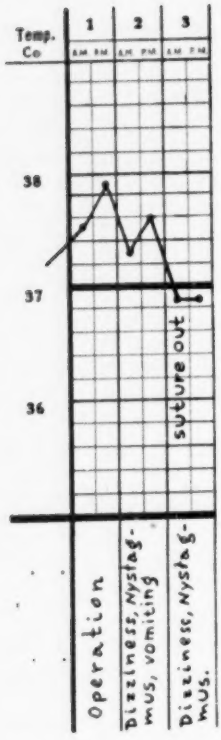

Fig. 3.—Temperature curve.

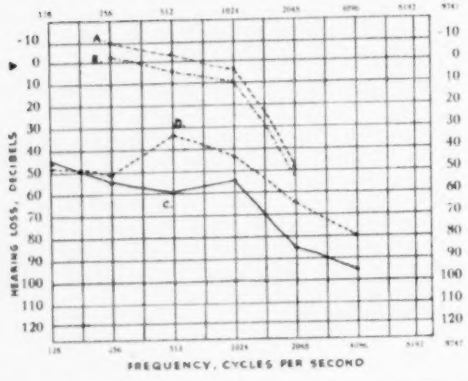

Fig. 4.—Preoperative and postoperative audiograms. *A*, hearing by bone conduction before operation; *B*, hearing by bone conduction eight days after operation; *C*, hearing by air conduction before operation; *D*, hearing by air conduction eight days after operation.

conduction a fall is noted at the 256 and 512 frequencies; this I ascribe to the temporary labyrinthitis present. This makes me believe that once labyrinthitis has disappeared hearing by bone conduction will increase.

An inadequate time has elapsed since the first operation to permit me to speak of any results or give any statistics. I hope, however, to do this in the future. I belive that the manner in which the fistula persists as well as the new mechanism of sound transmission are wholly different from what happens in Lempert's procedure, but I cannot say that my method is either better or worse than his as far as final results are concerned. It is, however, true that this technic opens up a new way of access to the middle ear and labyrinth, and it has many advantages over the technic now generally being used.

Hernán Cortés 29.

Case Reports

CARDIOSPASM WITH THORACIC STOMACH AND CONGENITAL SHORTENING OF THE ESOPHAGUS

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An elderly woman, at a period of mental stress, experienced sudden dysphagia. There were intermissions in which swallowing was possible, with a gradual return to normal. Some dyspepsia with flatulence remained. Several years later, again at a time of mental distress, the obstruction recurred. Swallowing of food caused a sense of pressure and fulness behind the sternum. There was no salivation. A roentgenogram after a barium swallow showed moderate dilatation of the esophagus to the level of the seventh thoracic vertebra (fig. 1). The stomach was not well outlined, as there was vomiting of the barium. Esophagoscopic examination demonstrated dilatation of the esophagus without residual food or ulceration and an obstruction at this level. A small Jackson bougie, 12 French, passed through the narrowing when relaxation with the anesthetic occurred. Before this, a filiform bougie would not pass. The transition from tight contraction to partial relaxation of the spasm was pronounced. A subsequent roentgenogram showed that the esophagectasia had diminished, the cardiac orifice was patent and the wall of the stomach was outlined as being partly within the thorax (fig. 2).

The spasm of the lower orifice of the esophagus was shown by the remittent nature of the dysphagia, varying from day to day and aggravated by psychogenic factors, and the disappearance of the spasm with deep anesthesia. (Injection of curare did not cause relaxation.)

This case poses the question as to the cause of this type of esophageal dilatation. The lower portion of the esophagus was, in this case, far removed from the crura of the diaphragm or the liver tunnel. Chevalier Jackson expressed the opinion that in such cases the diaphragmatic pinch-cock did not relax, and he gave the name of preventriculosis to the condition. Mosher concluded that inflammatory changes or hemorrhage around the esophagus, probably commencing in early life, lead to fibrosis, narrowing and kinking in the liver tunnel. Neither of these conditions were present in this patient. The symptoms, signs and esophagoscopic findings and results of dilatation were similar in all respects to cardiospasm in the normal situation of the esophagogastric junction.

It is noteworthy that stripping the sympathetic nerve fibers from the axis of the celiac artery—from which the lower end of the esophagus derives its supply—has been found to relieve the condition temporarily; and that division of the phrenic nerve, paralyzing the diaphragm, does not relieve the spasm. A high spinal anesthesia has, in some cases, completely cured the cardiospasm.

It appears, from the evidence presented and from the occurrence of the spasm at this abnormal level, that cardiospasm, or achalasia (Hurst), is due to spasm or lack of relaxation of the circular muscle of the esophagus at the junction of the esophagus and stomach, the cardiac sphincter.

12 Collins Street.

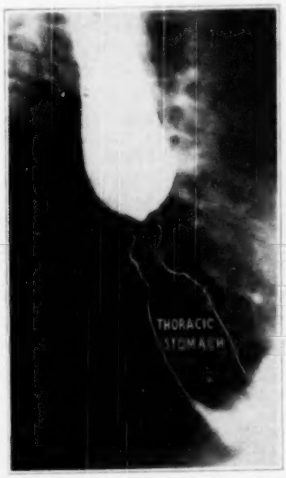

Fig. 1.—Prior to dilatation of cardiospasm.

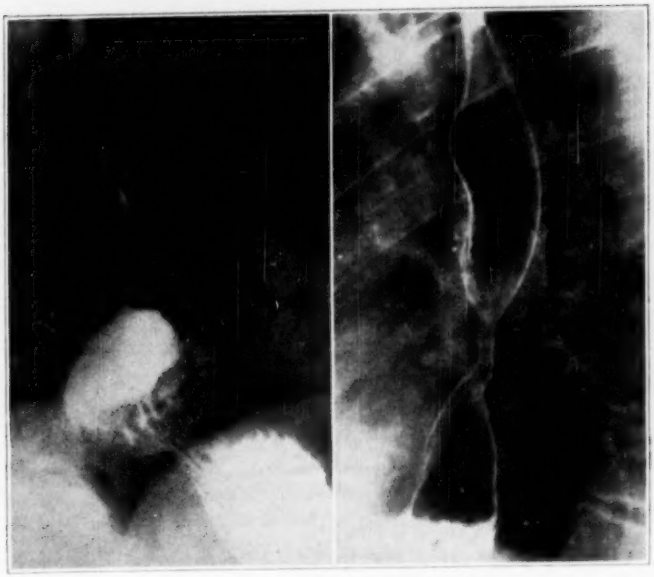

Fig. 2.—After dilatation of cardiospasm.

STENOSIS OF THE EXTERNAL AUDITORY CANAL

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AND
CHARLES F. WHITAKER, M.D.
CHICAGO

In the surgical correction of a stenosis of the osseous portion of the external auditory canal, it is undesirable to open into the mastoid cells because of the possibility of infection entering the cells during the later postoperative period. The surgical procedure used in the case to be reported illustrates a method of enlarging the external auditory canal without entering the mastoid cells or endangering the facial nerve deep in the posterorinferior aspect of the canal. It is readily possible to enlarge the canal by removing part of the bony meatus anteroinferiorly, thinning the tympanic plate anteriorly and removing the bone anterosuperiorly to the suture line (figure). If an attempt is made to enlarge the canal deep posteroinferiorly, there is danger of injuring the facial nerve in those occasional instances in which the facial canal lies more superficial than usual.

REPORT OF CASE

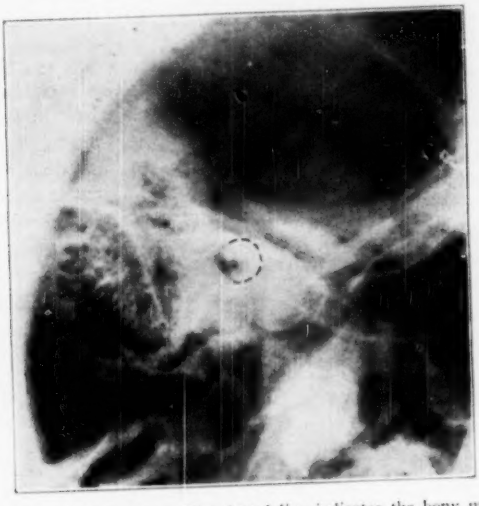
A woman aged 47 was first seen at the otolaryngology clinic of the Northwestern University Medical School on Feb. 11, 1947. She gave a history of having been in an automobile accident in 1941 and sustaining an injury to the left side of the mandible which resulted in ankylosis of the left temporomandibular articulation. In an operation performed elsewhere in December 1946, an attempt was made to free this ankylosis through an antetragal incision on the left side. The operation was unsuccessful, as the ankylosis was not relieved; in addition, the patient awoke from the operation with paralysis of the left facial nerve and deafness in the left ear.

On the initial examination by us in February 1947, almost complete cicatricial stenosis of the isthmus of the left external auditory meatus was observed. A small amount of purulent drainage pulsated through this opening. The tympanic membrane could not be visualized. Partial paralysis of the left facial nerve persisted. There was approximately a 50 decibel loss of hearing in the left ear. Roentgenograms showed extensive pneumatization of the left mastoid bone with no evidence of disease. Local treatment was ineffective in clearing up the infection. Because of the increasing stenosis of the canal, continued drainage and persistent pain, it was deemed advisable to relieve the stricture surgically and to explore the deeper part of the canal.

On April 30 the stenosis of the canal was exposed through a short superior endaural incision. The scar tissue narrowing the canal at the isthmus was excised. Deep to this point there was some debris, but the tympanic membrane was intact.

From the Department of Otolaryngology of Northwestern University Medical School.

The bony meatus was enlarged with a dental burr in all directions except postero-inferiorly, where there is danger of injury to the facial nerve. The denuded portion of the canal was lined with a split thickness skin graft obtained from the thigh. The graft was inserted into the canal by wrapping it around a short piece of rubber tubing which had been slit longitudinally in order that, owing to its elasticity, it might press the cut surface of the graft against the denuded portion of the canal. After the tube and graft had been inserted, the lumen of the tube



Enlargement of the meatus. The dotted line indicates the bony margin of the canal that was removed in order to provide a larger meatus. Enlargement of the meatus facilitated the insertion of the graft and lessened the likelihood that the stenosis would eventually recur.

was packed tightly with dry gauze dusted with sulfathiazole powder. The tube was removed on the twelfth postoperative day. The entire graft remained viable.

At the time of this report, one year after operation, the canal is widely patent and entirely free of infection. There has been almost complete recovery from the facial paralysis, but the deafness persists.

55 East Washington Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

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THE AVAILABLE literature of 1948 on otitis media and complications was reviewed and has been summarized under different headings. There were several very interesting trends noticed. The articles published in the United States on otic infections were much fewer than in the years past. This is partly because most such infections are now controlled so well by chemotherapy and penicillin and also because many otologists in the teaching centers are writing about the fenestration operation and its results.

Several European studies of otitis media and mastoiditis in infants were published. It is probable that poorer nutrition of infants (and mothers) would make this a greater problem in European countries than in the United States. No such papers have been published in the United States for some years.

PSYCHOSOMATIC MEDICINE IN OTOLOGY

Henner [1] reviews the psychosomatic basis for many symptoms projected to the ear, nose and throat—one of the few such articles yet to appear in a journal devoted to otolaryngology. He mentions that no specialty in medicine has been so mechanistic as otorhinolaryngology. When a patient is faced with a need to avoid or retreat from unpleasant situations, the psychosomatic stimuli often affect an organ that has been the site of previous pathologic change and has become a spot of least resistance. Henner states that "these patients require care as desperately as those who present acute surgical mastoiditis." "The attitude of theraputic nihilism . . . can result only in the patient's seeking another physician or in his failure to be benefited." He concludes that the otolaryngologist should treat these patients as long as their conflicts and symptoms do not impair their adjustments in relationships with others and gives Karl Menninger's six principles in counseling:

First, show the patient that you are interested in him.

Second, don't lie to him, don't give him placebos or joke with him about his symptoms, and don't promise him anything.

1. Henner, R.: Psychosomatic Components of Disease in Otolaryngology, Arch. Otolaryng. **47**:789-901 (June) 1948.

Third, listen to what he has to say, listen a long time, and listen many times, alone and without interruption.

Fourth, listen without censoriousness in word and expression, without rebuke, ridicule, or amusement. Absurd as aspects of them may be, your patient's maladjustments are not funny to him.

Fifth, give no advice, no treatment, and no opinion until you know what the patient is really unhappy about; then tell him that such unhappiness could be (not is) the cause of such symptoms.

Sixth, gradually help the patient to see the connection between his unhappiness and his symptoms and to realize that he must assume the responsibility for making such changes in his techniques or his environment as will be likely to give him greater peace. This, rather than allowing him to throw all responsibility on the doctor, is the rational and only permanently successful method of helping him.

[Although not primarily a paper on infections of the ear, this deals with a subject that none of us know enough about. Time used in reading this paper will be well spent.]

Alvarez [2] says every physician should know much about psychosomatic medicine, because without such knowledge he is likely each year to make hundreds of unhappy mistakes, order scores of futile operations and scare half to death hundreds of worrisome but organically sound persons. A large percentage of the patients one sees are ill mainly because of an inborn hereditary tendency to nervousness, worrisomeness, abnormal and painful ways of thinking and constitutional frailness. There is much need for greater skill in diagnosing functional troubles immediately from the history and greater skill in ignoring incidental conditions which cannot possibly be responsible for all the symptoms. One must learn when to suspect that a patient's main trouble is a nervous breakdown; one of the best signs of this is an inability to work comfortably if at all; another is inability to read, and such a patient usually cannot sleep.

In the discussion, Dr. Frank Allen, of Boston, said that when the usual patient is advised that an examination does not show organic disease he is relieved and grateful but the neurotic patient is apt to be disappointed, distrustful and unconvinced—and should be sent to the psychiatrist.

DIAGNOSIS

Mathiesen [3] reported the case of a patient in whom a draining postaural fistula developed after mastoidectomy. Drainage continued

2. Alvarez, W. C.: Psychosomatic Medicine That Every Physician Should Know, J. A. M. A. **135**:704-708 (Nov. 15) 1947.

3. Mathiesen, H. R.: Roentgen Examination of the Temporal Bone with Contrast Medium in Certain Ear Infections, Nord. med. **38**:1035-1037, 1948.

fifteen years. After injection of a contrast medium, a tract was shown roentgenologically to lead forward to a focus of infection in the malar bone.

BACTERIOLOGY

Brown, Krabek and Skiffington [4] tested the bactericidal effect of glycerite of hydrogen peroxide with urea in the laboratory. Three proprietary aural "antiseptics," auralgin,* otosmosan* and otomide,* were also tested. The glycerite of hydrogen peroxide with urea showed greater activity against all organisms tested than did the three proprietary preparations.

Hayes and Hall [5] made a careful bacteriologic study of 100 cases of otogenic infections, observing especially the effect of dibromosalicylaldehyde. It was found to be effective against both the gram-positive and the gram-negative organisms. The authors found that Pseudomonas aeruginosa (Bacillus pyocyaneus) was the commonest cause of chronic otitis media and external otitis. [Streptomycin was not available at the time this study was made. I have found that a powder of streptomycin mixed with about 4 parts of boric acid powder and blown into the ear canal is very effective against the gram-negative (and gram-positive) organisms if the infection is not too deeply seated.]

CHEMOTHERAPY AND BIOTHERAPY

Jersild,[6] of the department of epidemiology, University of Copenhagen, reported that since December 1945 a total of 2,000 patients with scarlet fever have been treated with penicillin in the Blegham Hospital. They found that patients treated less than six days would have recurrences of tonsillitis, otitis media, cervical adenitis, etc. If they were treated for six days, relapses were rare. In 1944 and 1945, in spite of energetic treatment with sulfanilamide, 21 patients (38 per cent) with scarlet fever otitis had to undergo mastoidectomy. Penicillin therapy was given to 56 patients admitted with scarlet fever otitis; only 2 required mastoidectomy, and in 1 of these mastoiditis was already present on admission. On follow-up examination none of the 2,000 patients treated with penicillin for six days was found to have any complication of scarlet fever otitis or nephritis. [This is an excellent report and bears out many other observations on the danger of stopping medications too soon; it also demonstrates the superiority of penicillin over the sulfonamides.]

4. Brown, E. A.; Krabek, W., and Skiffington, R.: Glycerite of Hydrogen Peroxide with Urea, Arch. Otolaryng. **48**:327-331 (Sept.) 1948.

5. Hayes, M. B., and Hall, C. F.: A Newer Concept of the Management of Otogenic Infection, Arch. Otolaryng. **47**:289-302 (March) 1948.

6. Jersild, T.: Penicillin Therapy in Scarlet Fever and Complicating Otitis, Lancet **1**:671-673 (May 1) 1948.

Abdeen,[7] of Alexandria, Egypt, treated 15 simple mastoidectomy wounds by packing them full of sulfanilamide powder moistened with a penicillin solution of 100,000 units in 1 or 2 cc. of isotonic sodium chloride solution. A short length of silver wire was left in the angle of the wound for drainage. All the ears were dry when dressed on the fifth day and remained dry. There were no reactions. The duration of drainage before operation and the preoperative treatment were not stated. The cases must have been advanced, because "operation revealed subperiosteal abscess in all cases—and extradural abscess was present in 9 cases." [Care is necessary in using large quantities of sulfonamides in mastoid cavities. Reactions with resulting slough in patients sensitive to sulfonamides have been reported. Many operators dust the mastoid cavities lightly with a sulfonamide powder and report good results.]

Young and Hall,[8] Edinburgh, Scotland, studied 115 cases of acute otitis media with special reference to hearing loss. In these cases there was an average of six days' treatment with penicillin, the inpatients receiving 100,000 units twice daily. Fifty-six patients had perforations (apparently from spontaneous rupture) on admission; 15 had mild otitis with redness but no bulging of the drum; 44 had bulging drums, but only 8 of these were incised. "There was no evidence to show that the cases in which incision was not done suffered any impairment of function (hearing loss) which might have been avoided by carrying out this procedure." In 6 cases simple mastoidectomy was required. The average ear was dry in seven or eight days. Normal hearing returned in eleven days on the average in the cases with suppuration and in nine days in those in which the drum did not perforate. In 8 cases there remained some hearing loss, and in only 1 was there a marked loss. In case of patients above 5 years of age audiometer tests were made; in younger patients loss was estimated by voice and whisper tests. In a comparable series reported in 1946 by Wilson in which the sulfonamides were used, there was somewhat slower healing but no higher percentage of loss of hearing. Young and Hall conclude that the busy practitioner who could not easily give daily treatments with penicillin may still rely on the sulfonamides, as their regular administration is preferable to irregular, inefficient use of penicillin. Finally they stress that penicillin and the sulfonamides are adjuncts to surgery and not substitutes and also that these treatments are applicable to cases of acute but not of chronic otitis media.

7. Abdeen, M. O.: Penicillin-Sulfanilamide Filling and Primary Suture in Simple Mastoidectomy, Arch. Otolaryng. **48**:332-336 (Sept.) 1948.

8. Young, A., and Hall, I. S.: Penicillin Treatment in Acute Suppurative Otitis Media with Special Reference to Long-Term Hearing, J. Laryng. & Otol. **62**:551-556 (Sept.) 1948.

Ramadier,[9] of Paris, reports 12 cases of otitis which from the onset were suspected of being cases of mastoiditis. Seven patients recovered without operation after administration of sulfonamides and penicillin; 4 recovered after mastoidectomy, and 1 died of encephalitis (proved by autopsy). All of the 5 on whom chemotherapy failed received intensive treatment, but the infections progressed in spite of adequate doses of the sulfonamides and penicillin.

ACUTE OTITIS MEDIA IN INFANTS

Hall and McGibbon [10] presented papers before the Royal Society of Medicine of England on the association of otitis media with gastroenteritis in infants. Miss Hall stated that during the years 1942-1947 she had been attached to six hospitals, in widely separated areas and had seen hundreds of such patients, 190 of whom required mastoid operations. She started out with the idea that the gastroenteritis was a result of the primary otitis media and mastoiditis but soon saw that the idea was untenable. By no means did all infants with gastroenteritis have otitis media, but in those who had mastoiditis with frank pus improvement after operation was immediate and often dramatic. McGibbon had operated on the mastoids of 67 of these infants; 31 of the patients (46.3 per cent) died. He was convinced that without operation mortality would have been over 90 per cent; many of the infants were moribund when seen. He stressed the necessity of early operation if myringotomy failed to improve the infant's condition within twenty-four hours. The operations were done with infiltration of procaine hydrochloride and usually took not more than five or ten minutes. Marriott's work published in 1930 is quoted.

Pearlman [11] reports a series of 37 cases of mastoiditis in infants occurring with the syndrome of diarrhea, vomiting and dehydration. He saw 500 cases over a ten-year period. Mastoiditis in infants may have an insidious onset and be easily overlooked. Any infant having severe gastrointestinal symptoms should have a careful check of the ears with the syndrome in mind. The mortality in the cases in which operation was done was 23.6 per cent.

Derham [12] reports 240 infants admitted to the Alder Hey Children's Hospital in England in 1946 with otitis media. There were also 191

9. Ramadier, J. A.: A Series of Disappointments with Sulfonamide-Penicillin Therapy in the Treatment of Acute Otitis Media, Semaine d. hôp. Paris (supp.) **23**:2579-2583 (Nov. 14) 1942.

10. Hall, W., and McGibbon, J. E. G.: Discussion on the Association of Otitis Media with Acute Non-Specific Gastro-Enteritis of Infants, J. Laryng. & Otol. **62**:396-418 (June) 1948.

11. Pearlman, S.: Mastoiditis in Infancy, Clin. Rep. Adelaide Children's Hosp. **1**:79-85 (May) 1948.

12. Derham, R.: Otitis Media in Infancy, Brit. M. J. 835 (May 1) 1948.

in whom otitis media developed after admission; 268 recovered, 163 (38 per cent) died. In spite of "barrier nursing technic" in the entire hospital, in many cases cross infections developed. Penicillin gave poor results, "possibly because of lack of sensitivity of the infecting organism." The mortality rate was about the same whether mastoidectomy was done or myringotomy relied on. [This problem has not been brought to our attention lately; it is well to remember that it exists.]

ACUTE OTITIS MEDIA AND MASTOIDITIS

Butler [13] observed 461 cases of purulent otitis media in students at an air base in 1942 to 1944. In 200 cases roentgen studies were made, and in nearly all of them there was some evidence of mastoiditis. In spite of the fact that sulfonamides were not given, mastoidectomy was required in only 2 cases. In only 6 cases were the patients transferred to other camps before dry ears were obtained. The average time elapsing before the ears became dry was two and one-half weeks, the limits being four days to six weeks. Butler concludes:

> It would seem that the morbidity of ear infections in recent years has paralleled the known decrease in morbidity of such conditions as measles and scarlet fever and that this, rather than the advent of the sulfonamides, accounts for the very happy outcome of the average ear infection.

[Many other writers have commented on the same tendency, but this should not excuse one from giving chemotherapy or biotherapy in case of otic infections of any severity.]

Fox and Minning [14] reviewed the cases of 18,659 patients with scarlet fever admitted to the Southview Isolation Hospital in Milwaukee from 1923 to 1945, inclusive. Otitis media developed in 9.38 per cent and mastoiditis in 0.76 per cent. Thirty per cent of the patients with scarlet fever (those with severer disease) received sulfonamides, and, although the incidence of otitis media did not drop very much, there was a definite decrease in the number of patients in whom mastoiditis developed. The incidence of otitis media was greatest in the ages of 5 years and under; 17.42 per cent of these children developed otitis media. [There are a number of charts illustrating the paper, which is carefully written.]

Bozer [15] cites Dean's figures, showing that in the United States in 1940 there was still 1 "ear" death in every 337 deaths and compares this with Paulson's figures of 1 "ear" death in every 303 deaths in Copen-

13. Butler, H.: Low Incidence of Mastoiditis in a Series of Cases Treated Without Sulfonamides, Ann. Otol., Rhin. & Laryng. **57**:992-998 (Dec.) 1948.

14. Fox, M. J., and Minning, V. L.: Incidence of Otitis Media and Mastoiditis in Scarlet Fever, Ann. Otol., Rhin. & Laryng. **57**:488-499 (June) 1948.

15. Bozer, H. E.: A Study of Surgical Mastoiditis Occurring in Children at the Buffalo Children's Hospital During the Years 1945 and 1946, New York State J. Med. **48**:183-186 (Jan. 15) 1948.

hagen over forty years ago. He analyzed 105 consecutive mastoidectomies occurring during the years 1945 and 1946 in the Buffalo Children's Hospital. The records showed that before admission only 43 per cent had any chemotherapy and of these 40 per cent had received inadequate therapy. Only 5 per cent were given penicillin, and only 1 received it for more than three days. In 26 of the 73 patients with spontaneous discharge rupture occurred after the third day; 13 had spontaneous rupture after the seventh day. "This in itself would seem to indicate either a considerable degree of indifference to the course of the disease or a lack of observation by the attending doctor." Bozer concludes that (1) earache is not considered seriously enough by patients and physicians; (2) paracentesis is not done promptly enough; (3) the infection is not considered sufficient cause for giving adequate chemotherapy; (4) there is not enough careful observation by trained otologists. In order to correct these faults he advises better teaching and supervision of medical students and interns.

Stuart Craig, in discussing this paper, mentioned, as have many others, that too much dependence is being placed on chemotherapy and biotherapy. Hoople commented that if early myringotomy had been done on each patient of this series, the majority could have been spared a mastoidectomy.

Watson [16] describes the old methods of caring for postoperative mastoid wounds and says that he now dusts the cavities lightly with penicillin and sulfathiazole powder, leaves a small drain in for two days and usually has a dry ear by twelve days. In severe infections penicillin is given intramuscularly and sulfonamides by mouth. In the discussion, L. Graham Brown mentions the danger of using chemotherapy and biotherapy as a substitute for surgery when pus has formed in the middle ear or mastoid cavity.

Vedrenne and Hays [17] report the case of a Negro boy of 11 years who was struck by a golf club over the left mastoid process. Five weeks later pain and purulent discharge came on during a cold. This subsided in five days, and two months later the boy was seen with a subperiosteal abscess. Roentgenograms of the mastoid did not show a fracture, but at operation a fracture line was found in the posterior wall of the canal extending down over the tip of the mastoid and through the antrum and inner table to the dura. An epidural abscess was found. The patient recovered with penicillin and sulfadiazine medication and had normal hearing afterward.

16. Watson, D.: Progress in Treatment of Mastoid Infection and Some of Its Complications, Proc. Roy. Soc. Med. **41**:155-164 (March) 1948.

17. Vedrenne, C. and Hays, J. R.: Acute Mastoiditis Secondary to Fracture of the Mastoid, New Orleans M. & S. J. **100**:504-506 (May) 1948.

ACUTE SEROUS OTITIS MEDIA

Tobey [18] expresses the belief that serous otitis media is a low grade infection caused probably by a specific virus. He has observed many cases that started with a painful throat with almost no redness of the pharynx, then after a few days a slight fulness of the ear would develop slowly into the typical symptoms of deafness with a feeling of fluid moving in the middle ear. His treatment is incision of the drum membrane followed by suction with a Siegel otoscope. He has not seen any cases in which the condition seemed to be due to allergy. Examination of the aspirated fluid showed no cells. He reports 2 cases in which mastoidectomy was done after the usual incisions failed to clear up the condition. Both patients showed edema of the mucous membrane and no necrosis, and both recovered with no more symptoms. In the discussion, Hoople mentioned 1 case in which the middle ear refilled three times in a few minutes after incision and removal of the fluid by suction. He assumed that the middle ear refilled from the mastoid. Dr. Westley Hunt spoke of 1 patient, a physician, on whom paracentesis was done eighty-three times in six years and another who had twenty-five incisions in four months. [This paper and the very interesting discussion should be read in its entirety by all otologists.]

CHRONIC OTITIS MEDIA

Gooch and Simonton [19] report the interesting case of a woman aged 47, a nurse and the wife of a physician. She had had intermittent scanty drainage from one ear since she had had scarlet fever at the age of 4 years. There was no drainage from the age of 19 to 42. Five years before admission she had occasional drainage with slight pain. She had numerous treatments by an otologist. Finally headaches and facial paralysis developed. Inspection showed only a tiny perforation in Schrapnell's membrane, which was covered with a thin crust of dried secretion. Roentgenograms showed erosion of the epitympanum. Radical mastoidectomy revealed extensive pathologic change; the horizontal canal was eroded; the ossicles were eroded, and the facial nerve was exposed. The facial nerve recovered after operation. [This case shows the necessity of routine check of this type of case by roentgenograms.]

Sellers [20] describes the various types of mastoiditis and the pathologic changes present. He states that he has seen several cases of cholesteatoma

18. Tobey, H. G.: Acute Catarrhal Otitis Media with Effusion, Tr. Am. Otol. Soc. **36**:51-71, 1948.

19. Gooch, J. O., and Simonton, K. M.: Diagnosis of Complications of Chronic Suppurative Otitis Media: Report of Case, Proc. Staff Meet., Mayo Clin. **22**:519-524 (Nov.) 1947.

20. Sellers, L. M.: Management of Chronic Suppurative Tympano-Mastoiditis, Texas State J. Med. **44**:378-381 (Sept.) 1948.

in patients with central perforations of the drum. For the past two years he has used the endaural approach for radical mastoidectomy and finds it more satisfactory than the postaural incision.

Dimant [21] raises the question as to whether the cholesteatoma is the result or the cause of the marginal perforation and chronic otitis media. Since occasional cases have been reported in which cholesteatoma has been present with apparently normal drums, he thinks it is possible that both conditions do occur. The object of his paper is to appeal to his colleagues to free themselves from the unproved hypothesis that chronic otitis media with marginal perforation is primary to and the cause of cholesteatoma. [Since cases of "primary" cholesteatoma are so rare, it is logical to suppose that the great majority of cholesteatomas are caused by chronic otitis media with marginal perforations. We should keep in mind that the other possibility does exist.]

Douglass [22] reports the use of nitrofurazone (furacin®) on 86 patients with chronic otitis media attending the outpatient department of Johns Hopkins Hospital. It was tried also on patients with chronic external otitis and a few with acute otitis media; 61 per cent were healed, 31 per cent improved and 5 per cent unimproved. It was found to be especially useful in patients with infections due to staphylococci, streptococci and diphtheroids. Fungi are not susceptible to the drug. Pseudomonas aeruginosa responded well.

Asherson [23] made a study of 479 cases of chronic otitis media seen in recruits during the last war in London. He made composite audiograms of the different types of ears seen. In the patients with chronic otorrhea the hearing loss averaged from 50 to 60 decibels and in those with cavities following radical mastoidectomy it averaged only slightly more. In patients with chronic attic disease with drainage (30 cases) it averaged about 25 to 30 decibels loss in the low and middle range and 60 decibels in the higher range. Of 29 patients who had had radical mastoidectomies 70 per cent had continued drainage. Eighteen patients operated on in private practice, whose after-care was in the hands of the operator, had dry cavities; their hearing loss was also about 60 per cent. [This study, in which special attention is given to the loss of hearing in different types of cases, shows the marked loss that occurs in chronic otitis media, whether or not operation is done. It should stimulate us to watch our patients with acute otitis media more closely and prevent this loss when possible.]

21. Dimant, M. D.: Cholesteatoma and Chronic Otitis, Arch. Otolaryng. **47**:581-589 (May) 1948.

22. Douglass, C. C.: The Use of Furacin in the Treatment of Aural Infections, Laryngoscope **58**:1274-1278 (Dec.) 1948.

23. Asherson, N.: The Radical Mastoid Operation: A Pre- and Post-Operative Audiometric Survey of Hearing Loss in Chronic Otorrhoea, J. Laryng. & Otol. **62**:568-602 (Sept.) 1948.

LATERAL SINUS THROMBOSIS

Novotny,[24] of Vienna, Austria, reported the case of a woman with symptoms of sepsis of ten days' duration from a discharging right ear. Mastoidectomy was done and the lateral sinus exposed; it was normal. In spite of penicillin and chemotherapy, metastatic foci appeared elsewhere in the body. Ligation of the right internal jugular vein was done. The next day the patient died of respiratory paralysis after a series of cerebral symptoms. Autopsy showed a thrombosis of the right lateral and the superior sagittal sinus and extreme cerebral edema. The ligated jugular vein was much larger than the other one; this may have helped to produce the edema and extension of the thrombus.

Harpman[25] reports 10 cases of sepsis of otogenic origin and 1 case following sinusitis. All these cases and 7 others not reported were observed since 1942. In only 3 of the cases reported did the patients receive penicillin; the others were apparently seen before penicillin was available. In 120 cases of otitis media in which penicillin was given the "results were improved by early treatment," but there was apt to be "incomplete resolution and recurrences." He concludes that "since the advent of present-day chemotherapy, surgery to the lateral sinus in otogenic sepsis should be limited to the drainage of pus and removal of loose infected clots without breaking down protective barriers." Ligation of the jugular vein should be done only in case there is extension of the infection down into the vein itself. He thinks that jugular ligation may bring on otitic hydrocephalus if the remaining jugular vein happens to be narrower than normal. [Fortunately this anatomic abnormality is extremely rare. If septic symptoms of chills and spiking fever persist after operation on the lateral sinus, the lesser of two dangers is ligation.]

Urbantschitsch[26] reported a fatal case of lateral sinus thrombosis in a 3 month old infant. The type III pneumococcus was found in pus in both mastoids and Staphylococcus aureus in the thrombosed lateral sinus.

Riccabona[27] reports the case of a woman of 70 who died suddenly after a short infection of the respiratory tract. Autopsy showed normal ear drums and a cholesteatoma lying on the temporal bone between the dura and the bone. There was considerable destruction of the internal auditory meatus and the semicircular canal from the apex to the arcuate

24. Novotny, O.: Thrombose im Sinus sagittalis superior nach Sinus-Jugularisoperation, Monatsschr. f. Ohrenh. **82**:327-329 (July) 1948.

25. Harpman, I. A.: Treatment of Otogenic Blood Stream Sepsis, J. Laryng. & Otol. **62**:11-21 (Jan.) 1948.

26. Urbantschitsch, V. E.: Mukosusotitis und Sinusthrombose mit Ikterus bei einem Säugling im dritten Lebensmonat, Monatsschr. f. Ohrenh. **81**:51-53 (Jan.) 1947.

27. Riccabona, H.: Angeborenes Cholesteatom, Monatsschr. f. Ohrenh. **81**:305-312 (June) 1947.

eminence. The cholesteatoma, which was judged to be congenital, extended to the mastoid antrum and epitympanic recess. Death was from meningitis apparently starting in this area. There were no signs of destruction of the drum and no infection of the middle ear cavity.

CHOLESTEATOMA

Lundgren [28] reports 2 cases of cholesteatoma and 2 other possible cases (2 proved by pathologic examination of tissue removed at operation and 2 which were reported as unspecified inflammation) in the petrous pyramid. These 4 cases were seen within one year, and Lundgren therefore decided that the condition is not so uncommon as has been supposed. In 1 case bleeding was so severe that the operation had to be done in three stages. The pathologic diagnosis based on the tissue removed was possible angioma. In all 4 cases the facial paralysis remained. Removal of the cholesteatomatous foci was done by destruction of the labyrinth. In 2 cases there was drainage of cerebrospinal fluid for two days, but there were no complications. The roentgenograms showing the lesions (Stenver's position) are reproduced.

Nilsson [29] reports the case of a man of 45 who had sustained a skull fracture ten years before. The fracture line went through the left petrous pyramid. There was bleeding from the left ear, but discharge did not develop until ten years later. There was found a small attic perforation with an otherwise normal drum. Operation showed a cholesteatoma the size of a pea with the old fracture line running toward it. The mastoid was normally pneumatized; the cells contained air and were lined with thin normal membrane. In 232 cases of cholesteatoma in which operation was done at his clinic since 1940 there was only 1 other case in which there was a normally pneumatized mastoid, and it may also have been traumatic. It was assumed that the epidermis got into the tympanic cavity through the fracture line and developed into cholesteatoma. Nilsson supports the Habermann-Bezold theory that cholesteatoma arises from ingrowth of epidermis of the external canal through perforations in Schrapnell's membrane.

MENINGITIS

In the three and one-half years from 1944 to 1947 Watson [16] observed 13 cases of meningitis with 2 deaths. This was a recovery rate of 85 per cent as compared with a 20 per cent rate in a seven year series before the introduction of sulfonamides.

28. Lundgren, N.: Cholesteatoma in the Petrous Bone Causing Progressive Facial Palsy, Acta oto-laryng. **36**:75-84, 1948.

29. Nilsson, G.: Attic Cholesteatoma Following Longitudinal Fracture of the Pyramid, Acta oto-laryng. **36**:85-91, 1948.

Merten [30] reported the case of a 61 year old man in whom after a slight earache for some weeks meningitis suddenly developed. Lateral sinus thrombosis was found, and the sinus and jugular vein were operated on. Near the labyrinth was found a fistula in the dura covered by fibrin. For twenty-eight days there was a discharge of 500 cc. of cerebrospinal fluid a day. Septic metatases developed, but with penicillin and sulfadiazine the patient recovered.

ENCEPHALITIS

Borries [31] goes over the literature on nonpurulent otogenous encephalitis from the first published case, in 1921 (written by himself), and states that the condition has at last been generally accepted and that a chapter on the subject is to be included in the international manual of the ear, edited by van Dishoeck and Gerlings. Two cases are reported which were seen in 1942. The patients had symptoms greatly resembling those of brain abscess but recovered without formation of pus; they were "discharged in wellbeing." [This is one of sixteen articles in a volume dedicated to Yrjö Meurman, of Helsinki, Finland. All the articles in this memorial volume are interesting, well written and well worth careful reading, as are all the numbers of the *Acta Otolaryngologica.*]

Kristensen [32] reports 3 cases of otitic encephalitis with symptoms of brain abscess. All 3 patients had pressure symptoms, such as coma, convulsions and vomiting, with stiffness of the neck. All of them were subjected to puncture of the brain with the expectation of finding a brain abscess. In the third case some tissue was aspirated. The microscopic examination showed extreme cerebral edema. In all of the cases the recovery was rapid and complete. Kristensen reasoned that an abscess if overlooked would not have cleared up so quickly. The cases were seen in 1940, 1941 and 1943, and the patients were aged 10, 48 and 9 years.

Hutton [33] reported the case of a girl of 14 who had had a draining ear since 3 years of age. After the appearance of early meningeal symptoms a mastoidectomy was done. The brain was explored twice for abscess, but none was found. Some pus was found inside the dura in the temporal area, and soft inflamed brain tissue was seen. With

30. Merten, A.: Meningitis Caused by Pneumococcus Type 3 with Cerebro-Spinal Fluid Drainage for Twenty-Eight Days, Monatsschr. f. Ohrenh. **82**:274-279 (June) 1948.

31. Borries, A. V. T.: A Contribution to the Clinic of Nonpurulent Otogenous Encephalitis, Acta oto-laryng., 1948, supp. 67, pp. 13-19.

32. Kristensen, H. K.: Sur les encéphalites otogenes, Acta oto-laryng. **36**:151-159, 1948.

33. Hutton, T. B.: Clinical Record of Response of Observed Encephalitis to Penicillin and Chemotherapy, J. Laryng. & Otol. **62**:603-607 (Sept.) 1948.

penicillin intramuscularly and intrathecally and sulfadiazine the recovery was complete, with no residual symptoms.

[These three articles confirm the opinion that encephalitis is not necessarily a stage of brain abscess any more than pneumonia is necessarily a stage in lung abscess.]

LABYRINTHITIS

Gadolin [34] reports the unusual case of a 21 year old soldier in whom facial paralysis, labyrinthine vertigo and vertical nystagmus developed after six weeks of otitis media. At operation the antrum did not show infection. Below the antrum was a pea-sized cavity filled with pus and granulations. This small focus had burrowed backward into the base of the labyrinth and penetrated the facial ridge into the tympanic cavity. Pus forming at the base of the labyrinth drained along the tract into the middle ear without going through the antrum. The patient recovered after operation, but he had persistently positive results of fistula tests.

Watson,[35] in his presidential address before the Section of Otology of the Royal Society of Medicine, discussed labyrinthitis and several other subjects. From 1922 to 1925 he tested all patients with chronic mastoiditis with caloric and rotation tests. He operated on 3 patients with signs of labyrinthitis, and all 3 died of meningitis. He found that his colleague had had no deaths and had not tested any patient with labyrinthitis. In the succeeding twenty-one years he did not test or operate on his patients with labyrinthitis and has had no more deaths. He concluded that even when symptoms were severe the infection might be only serous labyrinthitis and the patient would recover without intervention on the labyrinth. Meningitis is not, as a rule, caused by labyrinthitis, but it is always a primary complication of mastoiditis. [Watson's comments on meningitis and other subjects will be given under the proper headings.] In the discussion, Cawthorne and others of the audience agreed with Watson's opinions. In closing, Watson compared labyrinth tests during labyrinthitis to tests on the heart. "Would a clinician test a patient with a temperature of 102 F. whom he thought was developing endocarditis by asking him to run upstairs and down again?"

Van Egmond and Jongkees,[36] Utrecht, Netherlands, report 74 cases of labyrinthitis with 6 deaths. No operation on the labyrinth was done,

34. Gadolin, H. R.: An Uncommon Syndrome—Acute Otitis, Facial Paralysis, Labyrinthine Vertigo, Vertical Compression Nystagmus and the Underlying Patho-Anatomy, Acta oto-laryng. **67**:24-27 (Feb.) 1948.

35. Watson, D.: Progress in Treatment of Mastoid Infection and Some of Its Complications: President's Address, J. Laryng. & Otol. **62**:465-476 (July) 1948.

36. van Egmond, A. A. J., and Jongkees, L. B. W.: Treatment of Inflammations of the Inner Ear by Sulfa-Drugs, J. Laryng. & Otol. **62**:447-459 (July) 1948.

and the authors conclude that operations on the labyrinth are never indicated. They cite the discussion at the meeting of the Royal Society of Medicine, Section of Otology, in 1944 in which D. Watson and Norton Canfield advised against caloric or turning tests in labyrinthitis. In the same discussion, Stirk Adams gave an example of meningitis developing after a caloric examination. Egmond and Jonkees will publish soon a case of a "sound vestibular organ considerably damaged by rotation tests," but they do not consider that a caloric test done in the presence of vestibular symptoms is dangerous. They advise radical mastoidectomy for chronic infection or simple mastoidectomy for acute infection when labyrinthitis develops, but they leave the lateral wall of the labyrinth untouched and consider scraping off of granulations as dangerous as any operation on the labyrinth. They cite Alexander's statement that a closed labyrinthitis is a disease without danger but labyrinthectomy is a dangerous operation.

BRAIN ABSCESS

Gerlings [37] reports 3 cases of metastatic brain abscess with autopsy findings seen in the University Hospital at Amsterdam. Metastatic brain abscesses are rare as compared to those that are formed by extension of thrombophlebitic processes and direct extension to the nearby temporal lobe. He was able to find 30 cases of metastatic cerebral abscesses published in the literature up to 1939. One of his patients was seen in 1936, 1 in 1941 and 1 in 1943. The last two patients received "prontosil," an early sulfonamide drug.

McKenzie [38] reported a case of brain abscess of otogenic origin in which roentgenograms showed a gas-filled cavity with a fluid level. The gas escaped under pressure when a needle was inserted. About 5 cc. of straw-colored fluid was withdrawn which was sterile. The fluid was replaced by thorotrast,® which shows well in the roentgenograms. [Whether the patient survived is not mentioned.]

Piquet [39] (France) has seen 19 patients with cerebellar abscess; 11 of them recovered. The most frequent symptom was intense, persistent headache which is localized in the affected temporal region. Nystagmus of wide amplitude with movement toward the affected side was next in importance. The symptoms are frequently slight and may not be noticed unless the possibility of such an abscess is kept in mind. Operation should be done early, before the stage of mental confusion is reached. The prognosis is good if the abscess is superficial.

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37. Gerlings, P. G.: Metastatic Brain Abscesses and Encephalitis of Otitic Origin, J. Laryng. & Otol. **62**:525-532 (Aug.) 1948.

38. McKenzie, W.: A Case of Gas-Filled Abscess in the Temporal Lobe, J. Laryng. & Otol. **62**:460 (July) 1948.

39. Piquet, J.: Little Known Symptoms of Otogenous Cerebellar Abscesses, Presse méd. **56**:270-271 (April 10) 1948.

Asherson [40] reports 5 cases of cerebellar abscess in detail and urges careful observation for early symptoms. Early diagnosis and early operation resulted in cures in his own 4 cases. In the fifth case (not his own) the condition was diagnosed early, but delay in operation resulted in death. Otogenic cerebellar abscesses may develop to huge size with very few symptoms other than a frontal or occipital headache and a slight stiffness of the neck. There may be only slight vomiting. The key to early diagnosis rests in spinal tap with finding of increased pressure and an increase in the cell count. A very practical point was the advice to question the adult coming for treatment for a long neglected draining ear: Why do you come *now*? "It is possibly because there is some subtle difference of which the patient has now become aware—the harbingers of the impending intracranial complication."

PETROSITIS

Fleishmann,[41] Budapest, has prepared an extensive article on silent petrositis. He calls it secondary osteomyelitis of the pyramid. He reports 7 cases in patients whose ages ran from 25 to 75. There were three deaths. In 5 cases the Ramadier operation was done; 2 of the patients recovered, and 3 died. In only one of the four operations was he able to say that pus was found and evacuated by the Ramadier procedure. He concludes that "secondary osteomyelitis of the petrous pyramid is the silent form of petrositis" and can be found only by systematic and repeated roentgenologic studies of the petrous pyramid. It is characterized by its lack of characteristic symptoms, such as paralysis of the sixth nerve with resulting double vision. The most constant symptom was severe headache. He advises the Ramadier operation, which is the anterior approach between the carotid artery and the eustachian tube. [Fortunately there are fewer cases of petrositis seen among patients treated by chemotherapy and biotherapy. It seems hardly necessary to adopt the new name of "secondary osteomyelitis of the petrous pyramid," but it is well to remember that fatal petrositis may exist without the classic symptoms of paralysis of the sixth nerve and pain referred to the eye along the branches of the fifth nerve.]

FACIAL PARALYSIS

Farrior [42] reports 6 cases of facial paralysis, in 2 of which the operation of nerve graft was required. Good results were obtained in both cases, the patients obtaining good motion of the face, one starting

40. Asherson, N.: Cerebellar Abscess, Acute, Otogenic, J. Laryng. & Otol. **62**:278-307 (May) 1948.

41. Fleishmann, L.: Ueber sekundäre Osteomyelitis der Pyramidenspitze, Acta oto-laryng., 1948, supp. 66, pp. 1-48.

42. Farrior, J. B.: Facial Paralysis in Otology, South. M. J. **41**:348-357 (April) 1948.

in 5 months and the other in 6 months. Another case resulted from a fracture of the skull. At operation bony spicules were removed from contact with the nerve, and recovery occurred, beginning two months later. There are a summary of the literature and several illustrations showing the technique of nerve graft.

TUBERCULOSIS OF THE MASTOID AND MIDDLE EAR

Greif and Gould [43] report 3 cases of otitis media and 1 of bilateral mastoiditis of tuberculous origin. One of the patients with otitis media showed no improvement with the use of streptomycin. The other 3 had healing with its use. In the patient with bilateral mastoid involvement a facial paralysis developed on one side and a mastoidectomy revealed extensive destruction of the cells with destruction of the dural plate. The facial paralysis disappeared within a week of the operation. In spite of streptomycin generally, the other ear did not heal and a simple mastoid operation was done on the second ear. Both ears healed with marked loss of hearing. In the case of otitis media which did not respond to streptomycin the tubercle bacilli were found by laboratory study to grow in media with a concentration of streptomycin greater than 10 units per cubic centimeter. This showed a resistant organism. The authors had good results in 7 cases of tuberculous laryngitis and 1 with involvement of the nasal septum.

Adams [44] reports 2 cases of severe tuberculous otitis media and mastoiditis, both occurring in patients with severe pulmonary tuberculosis. In the first case, after draining for two years, the ear healed without operation and remained dry until the patient's death, three years later. She died of pulmonary tuberculosis in 1941. In the second case severe tuberculous otitis developed with roentgen evidence of mastoiditis. With the administration of streptomycin the ear became dry in one month and has remained dry. There was very little loss of hearing.

OTITIC HYDROCEPHALUS

McKenzie [45] (London) reports the case of a 12 year old girl with otitic hydrocephalus. She had had intermittent otitis media. On admission she had a discharging ear of eight days' duration. Symptoms of intracranial pressure developed ten days later in spite of penicillin. Two weeks later mastoidectomy was done, but infection was not found. The cerebrospinal fluid was normal, but the pressure was as high as

43. Greif, J. L., and Gould, J. W.: Tuberculosis of Ear, Nose and Throat Treated with Streptomycin. Arch. Otolaryng. **48**:209-220 (Aug.) 1948.

44. Adams, J. G.: Tuberculosis of the Middle Ear and the Temporal Bone. Ann. Otol., Rhin. & Laryng. **57**:980-983 (Dec.) 1948.

45. McKenzie, W.: A Case of Otitic Hydrocephalus, J. Otol. & Laryng. **62**:391-393 (June) 1948.

350 mm. at one time. The child was improving at the time of the report. McKenzie commented that the term otitic hydrocephalus is misleading since internal hydrocephalus is never seen and only 1 or 2 cases of external hydrocephalus have been encountered. Also by no means do all the patients have infected ears. He accepts Symonds' summary of the disease, which is as follows:

1. Children and adolescents are almost always affected.
2. Between attacks of headache the patient feels and looks well.
3. Papilledema is constant, usually with measurable swelling.
4. There are no localizing signs but a 6th nerve paresis on the side of the lesion is often present.
5. Lumbar puncture shows a clear cerebrospinal fluid with increase of pressure of over 300 mm.

The pathologic basis is generally agreed to be an obstruction to the venous circulation of the brain by thrombosis. No treatment is effective but the patients usually recover slowly.

GRADENIGO'S SYNDROME

Horowitz [46] reviews the symptoms of Gradenigo's syndrome and discusses the pathology. In 1 of his 2 patients otitic hydrocephalus developed, with an intracranial pressure producing a papilledema of 5 D. without affecting the sight. Both patients recovered after mastoidectomy. An operation for petrositis was not necessary in either case.

TECHNIC

Tumarkin [47] reports 300 cases of chronic otitis media in which operation was done by his "transmeatal atticoantrotomy" technic. There were no complications or deaths in the 300 cases; transient facial palsy occurred in 3 cases. Fifty recent consecutive cases were reviewed. Replies were received from 42 patients; in 38 the cavity was healed; in 9 of these the middle ear remained moist; 21 of the 42 reported improved hearing, 19 no change, and 2 had more loss. The operation is done through an aural speculum and the field is rarely more than 8 mm. across. He quotes Thies as having operated in 1,500 cases without a fatality. The operation takes from twenty to forty minutes, and the patient goes home in five days. (The instruments used were described in another article as tiny gouges 2 and 3 mm. wide.) The incision begins at Schrapnell's membrane, goes upward and then curves backward and downward. A skin flap is elevated and rolled down onto the

46. Horowitz, S.: Gradenigo's Syndrome and Report of Two Cases, J. Laryng. & Otol. **62**:639-647 (Oct.) 1948.

47. Tumarkin, A.: Mastoid Surgery—Old and New, Lancet **1**:708-711 (May 8) 1948.

floor of the canal during the operation. After adequate removal of bone, including the ossicles if they are diseased, the skin flap is rolled down over the raw surfaces and the cavity packed with a penicillin preparation. [The modified radical operation would probably be better and safer in the hands of the average operator.]

Norup [48] reports that since 1942 he has done simple mastoidectomy in 60 cases of chronic otitis media, packed the postoperative wound with sulfathiazole powder and completely closed off the postauricular incision. In all cases there had been draining for at least three months and in 67 per cent for more than two years. In 28 cases there were marginal perforations. In 16 cases there was cholesteatoma, and "in 8 of these cases atticoantrotomy was performed." Healing with dry ears was obtained in 67 per cent of the cases, and in 33 per cent chronic discharge continued. In a control group treated by the same type of operation but in which packing with iodoform gauze was done there remained a chronic discharge in 58 per cent of the cases. The hearing loss is stressed as being less in the group in which primary suture was used than in the control group. [It is hard to understand why these cases were not done by the modified radical technic which would give the patient the safety of an open cavity with preservation of hearing.]

MISCELLANEOUS

Cherubino [49] reported an interesting case of severe spontaneous bleeding from the internal carotid artery following chronic suppuration of the ear. He found 36 cases reported in the literature, with 10 survivals. Ligation of the internal carotid artery was advised if packing of the ear did not control the bleeding.

Ochs [50] reports the very unusual case of a man who fell while smoking a pipe. The stem of the pipe penetrated the middle ear from the mouth, broke off and remained in the middle ear. There was immediate complete deafness and paralysis of the right side of the face. After removal of the fragments by a radical mastoidectomy there was a return of most of the function of the facial nerve, but no hearing.

48. Norup, S.: Treatment of Otitis Temporalis Chronica by Simple Chiselling, Sulfathiazole Plombage and Primary Suture, Acta oto-laryng. **36**:7-16, 1948.

49. Cherubino, M.: Treatment of Bleeding from the Internal Carotid Artery into the Petrous Canal, Boll. d. mal. d. orecchio, d. gola, d. naso **65**:185-194 (Sept.-Oct.) 1947.

50. Ochs, I. L.: Foreign Body of the Middle Ear: Case Report, Arch. Otolaryng. **47**:670-672 (May) 1948.

News and Comment

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

The officers of the Indianapolis Ophthalmological and Otolaryngological Society for 1949-1950 are Dr. Alan L. Sparks, president; Dr. Edwin D. Dyar, vice president, and Dr. J. Lawrence Sims, secretary-treasurer.

HYGEIA BECOMES TODAY'S HEALTH

The trustees of the American Medical Association have authorized the renaming of the Association's health magazine *Hygeia*. The new name will be *Today's Health*. The change will be made with the issue of March 1950.

CORRECTION

In the article by Dr. Oscar J. Becker, "Surgical Correction of the Abnormally Protruding Ear," in the November 1949 issue of the ARCHIVES, page 541, the last two words of the legend to section *C* of figure 3 should be fissura antitragohelicina. Also, on page 558, the concentration of epinephrine hydrochloride (second paragraph under "Operative Technic") should be 1:50,000.

Abstracts from Current Literature

Ear

REHABILITATION OF HARD OF HEARING CHILDREN: A SUMMARY OF THE LAWS AND AN ANALYSIS OF THE SCHOOL TESTING PROGRAM IN NEW YORK STATE. GEORGE J. RIGHTER and PAULINE K. WINKLER, New York State J. Med. **48**:83 (Jan. 1) 1948.

Total deafness is usually discovered early in childhood. Education, including tuition and maintenance, for the unfortunate patients is provided by six institutions in the Empire State. Those who are hard of hearing (not totally deaf), state the authors, "can be completely rehabilitated if all means at our disposal are used." Education and the application of existing laws facilitate the desired end.

A summary of existing laws indicates that four acts are in operation: 1. The Reporting Act provides that all physicians, nurses, parents and guardians must report hearing impairment in children under 6 to the State Commission of Health in order that the child may be properly oriented and cared for. 2. The Audiometer Act provides that all school children must have an audiometer test at least once each year. Thus, poor hearing may be discovered promptly. The state supplies a booklet of detailed instructions for testers. If there is a 9 decibel loss a test with the pure tone audiometer is required; 10 decibel loss recorded with the pure tone audiometer calls for examination by an otologist, and a 20 decibel loss is reportable to the Commission of Health. This applies to all cities save New York, Buffalo and Rochester! 3. The Education Act provides for transportation to school, a visiting teacher in the home and special classes. 4. The Rehabilitation Act defines a handicapped person as one who is incapacitated, from whatever cause, for earning a living. Such a person may become a ward of the state if he is not too old or helpless, mentally diseased or under 14 (the employment age). All other persons over 14 who have a physical defect because of which they are totally or partially disabled may apply for aid or advice. The Advisory Commission may arrange for treatment leading to rehabilitation except for those who are eligible under the Workmen's Compensation Law. The state may provide the cost of maintenance during actual training and transportation, but the amount must not exceed $15 a week! Training courses under employers are paid for by the state, and books and tools may be so provided.

Statistics of the Conservation of Hearing Department of the Division of Health and Physical Education of the State Education Department gave the total number of students, exclusive of those in the three cities mentioned, as 707,767 during the school year of 1944 and 1945. Of these students, 65.3 per cent received the audiometer test in groups. Pupils recommended for special training numbered 91.2 per cent, but not all took advantage of the opportunity. There are Conservation of Hearing classes in four cities: Albany, Schenectady, Gloversville and Syracuse. The charge for tuition is set for nonresident students by the local school superintendent. In addition to these four classes, twenty-eight communities offer lip reading classes. All but six of these are in the metropolitan area, including Long Island and Westchester County. The six others are located in Auburn, Binghamton, Ithaca, Olean, Utica and Watertown. Statistics for the period from 1944 to 1945 show that 1 child in every 53 of the 462,304 tested had a loss of 15 decibels or more, that 1 in 500 was recommended for special education and that

1 of every 907 received it. It is notable that the Catskill and Adirondack areas have no rehabilitation facilities whatsoever.

Certain conservation of hearing clinics in approved hospitals serve to assist in the classification of children and in their direction toward the sources from which they may expect help. Two types of educational service are still needed: (1) conservation of hearing classes in public schools, with full time teachers in large cities and towns, and (2) lip reading and speech correction classes in areas where there are no conservation of hearing classes. There is a need for specially qualified teachers for the deafened, and colleges and universities throughout the state have a responsibility to offer training for such teachers. VOORHEES, New York.

THE CONSERVATION OF HEARING PROGRAM IN NEW YORK STATE. GORDON D. HOOPLE, New York State J. Med. **48**:169 (Jan. 15) 1948.

This paper was read before meeting of the Medical Society of the State of New York at Buffalo in May 1947. It is a plea for the support of otolaryngologists in setting up centers to aid deafened persons. Sending a patient to a hearing aid company to get rid of him is decried. It is the specialist's responsibility to see that a suitable aid is fitted and supplemented by auricular training and lip reading. Reference is made to the "practical program" offered by the American Academy of Ophthalmology and Otolaryngology, but Hoople states that he does not find it entirely satisfactory. The otologist is often too busy to see his deafened patient through; therefore, centers may help to solve the problem. Aural rehabilitation centers as instituted by the Army and Navy are commended, and it is said that 90 per cent of those patients questioned in 1945 were still wearing their aids with satisfaction after more than a year. It is assumed that civilian centers might produce similar results. Courses in universities are educating personnel in audiometry, speech training and correction, reading and the fitting of hearing aids. Data concerning equipment for centers, including radium, are given. New York, Albany, Rochester and Syracuse have centers, but "a large part of the populace (of the state) is unserved." Public funds must be provided to finance many centers in all parts of the state; "otolaryngologists should band together" to see that the program is adopted. It is suggested that these centers be called "Conservation of Hearing Centers."

VOORHEES, New York.

OTOSCLEROSIS: ITS PATHOLOGY AND TREATMENT. FRANZ ALTMANN and DEGRAAF WOODMAN, New York State J. Med. **48**:173 (Jan. 15) 1948.

The diagnosis of otosclerosis is usually based on insidious and progressive deafness, often associated with tinnitus and dizziness. Office treatment is useless. Surgery is indicated if an aid is ineffective and if nerve degeneration has not progressed. In any case, careful selection is in order. The cause of otosclerosis is unknown, and anything one may do is merely palliative, including fenestration, which these authors say is "the greatest achievement in this special field of otology since the introduction of the caloric vestibular tests by Bárány about forty years ago." Fenestration not only restores the hearing range, at least partially, but it has led to further study of the physiology of hearing. Out of all this cooperative effort among various workers there may yet come relief from other forms of deafness still regarded as incurable.

The authors state the belief that the frequent coincidence of pregnancy and otosclerosis has little eugenic significance, for "only 15 per cent of the persons afflicted with otosclerosis show reduced hearing; the other 85 per cent who have normal hearing remain undiagnosed and may nevertheless transmit the disease. . . . There is no hope . . . of eradicating the disease by even the most drastic eugenic measures."

VOORHEES, New York.

OTITIS EXTERNA GRANULOSA. NORMAN A. PUNT, Brit. M. J. **1**:989 (June 4) 1949.

Thirteen patients with otitis externa granulosa were seen while the author was otologist to the British forces in Austria and Northern Italy. The granulations appeared in cases of suppurative meatitis in which the condition was severe and had been neglected.

The important point on which emphasis is laid is the likelihood that such a condition will be diagnosed as chronic suppurative otitis media by the unwary physician. Differential diagnosis was made after careful cleansing of the meatus, using peanut oil or olive oil on wool mops, and removal of granulations with forceps and/or a silver nitrate "bead" fused onto a probe. The disease responded rapidly to the cleansing. An intact drumhead with normal landmarks could usually be seen at the second or third visit. Other points of differentiation (which was facilitated by the gentle use of a probe and of Siegle's pneumatic speculum) were the foul odor of the discharge and the absence of mucus; pulsation and the presence of a meatal fistula indicated a form of chronic suppurative otitis media.

PUNT, London, England.

ARACHNOIDITIS OF OTITIC ORIGIN. STÉPHANE KYLAR, Pract. oto-rhino-laryng. **9**:57, 1947.

Kylar discusses arachnoiditis in its various forms. Its frequency is difficult to determine since the symptoms are often insidious and transitory and may occur at any time after the onset of acute otitis or of a suppurative infection of the mastoid process. Propagation from the middle ear may take place by the cellular route, the infection being transmitted directly from the mastoid cells; by the larbyrinthine route, the infection entering the labyrinth at some point of lessened resistance (either by way of a fistula or by way of the preformed anatomic dehiscence), or by the vascular route, being due to an infection of either the lateral sinus or the superior petrosal sinus.

There are four principal forms: (1) adhesive or plastic arachnoiditis, occurring frequently about the optic chiasm or about the trunk of the eighth nerve; (2) the cystic form, occurring in the subarachnoid space, usually in the posterior cranial fossa; (3) the form of internal hydrocephalus, due to inflammatory occlusion of one or more of the orifices of the ventricles of the aqueduct of Sylvius or of the fourth ventricles, and (4) the form of generalized hypertension, in which the cerebrospinal fluid circulates freely in the subarachnoid space. This process may be due either to hypersecretion of the fluid or to a decrease in absorption due to some inflammatory reaction about the choroid plexus. The symptoms are usually clearcut, and the condition is part and parcel of cerebral hypertension. The intensity, frequency and origin depend on the underlying pathologic factors. Headaches are the commonest symptom. The pain may be localized at one point or may be diffuse. It is often paroxysmal, appearing most intense in the morning

and lessening toward evening. Vomiting often accompanies these headaches. Ocular changes occasionally appear, but these depend on the extent of intracranial hypertension. They include arterial changes and fixation of the pupil. In general, the symptom complex includes cerebellar and vestibular abnormalities. Interference with the normal responses of the labyrinth in rotation and in the caloric and galvanic tests depends greatly on the underlying pathologic factors. The different clinical forms of arachnoiditis are described as (1) arachnoiditis in the region of the cerebellopontile angle and the auditory canal, (2) arachnoiditis of the large cistern, (3) generalized arachnoiditis of the posterior cerebral fossa and (4) precerebellar and prepontile arachnoiditis.

Therapy is recommended as follows: When there is frank otorrhea, complete mastoidectomy should be performed to eliminate every avenue of infection. To counteract hypertensive syndromes, intravenous injections of hypertonic solutions of magnesium sulfate, dextrose or calcium are of value. In addition, the oral administration of ammonium chloride and a salt-free diet should be instituted. Roentgen therapy has given good results in diffuse arachnoiditis. However, if the therapeutic procedures outlined are not satisfactory the case should be referred to a neurosurgeon. A ventriculogram and an encephalogram are extremely valuable in some cases. The injection of penicillin has helped, but a neurosurgeon may finally have to perform decompression or remove the cyst. In the handling of these patients, the otologist, the ophthalmologist, the neurologist and the neurosurgeon must all be consulted to help solve the problem. In conclusion, the author states: "Every otitis media can be the origin of an arachnoiditis. The diagnosis is often difficult, as the clinical picture is by no means constant. A slow course with a crisis-like intermission, after which all symptoms have more or less disappeared is characteristic of an arachnoiditis." The two commonest symptoms are headaches and vertigo. On the other hand, one must remember that there are no pathognomonic signs in this disease.

PERSKY. Philadelphia.

Larynx

BRONCHIECTASIS. LAWRENCE MISCALL, New York State J. Med. **49**:1070 (May 1) 1949.

Miscall analyzes 146 cases of clinical bronchiectasis recorded in the thoracic service at Bellevue Hospital. Ten per cent of the patients were under 20, and 62 per cent were under 50. There were instances of constant cough; copious, foul sputum; recurrent pneumonia; hemoptysis; abscess, and empyema. Of the 146 patients, only 44 were operated on.

The lack of attempts at curative therapy contributes to marked deterioration of the entire system. Medical care calls for rest in bed, adequate diet reenforced with proteins, vitamins and minerals and intravenous administration of blood to overcome the anemia and asthenia. Sinusitis and oral sepsis should be diagnosed and overcome. Bronchoscopy should be routinely practiced for diagnosis and treatment. The removal of secretions by suction brings relief, and dried exudate, granulations, foreign bodies, etc., may be found and treatment carried out accordingly. Tumors and stenosis usually call for a change of treatment.

Cardiorespiratory considerations influence operability. The younger patients usually present more operable conditions than the older ones. Surgical intervention should be carried out promptly once the matter of operability is settled. The best time for the operation is when the sputum is reduced in quantity and its quality is less dangerous. In some cases, preliminary drainage and secondary excision

offer more than the one stage procedure with extensive removal of lung substance. The intrahilar dissection technic is recommended. The safety of lobectomy or segmental resection is stressed, the mortality being only 2 per cent.

VOORHEES, New York.

THE ROLE OF BRONCHOSCOPY IN THE PREOPERATIVE DIAGNOSIS OF CARCINOMA OF THE LUNG. ADRIAN LAMBERT, N. Y. State J. Med. **49**:1173 (May 15) 1949.

Early diagnosis and prompt surgery now offer a better prognosis in carcinoma of the lung than heretofore. Bronchoscopic examination is only a minor aid, since many tumors, especially those situated peripherally, cannot be visualized through a direct tube. Those in a central location can be partially visualized, but proximity to important organs makes biopsy somewhat hazardous. Certain points should be noted by the bronchoscopist: (1) Lateral compression of the trachea suggests the presence of a mass of some kind; (2) widening of the carina may be due to enlargement of a lymph node, which is often cancerous; (3) angulation of the main bronchus may signify atelectasis from compression of the tumor; (4) longitudinal striae in a bronchus may be due to external compression, and (5) bleeding is always suggestive of a cancerous growth somewhere in the lung. The Papanicolaou stain helps in diagnosing the malignancy of tumor cells. These may be found in the "washings," since tumors shed cells as they grow. A single malignant cell may set the diagnosis, but it must be certified by an expert cytopathologist. Exploratory thoracotomy is permissible since it determines the diagnosis. Whereas bronchoscopy was formerly determinative in only about 58 per cent of cases, with the newer technic it may be determinative in 90 per cent. Biopsy and washing of cells are in the first rank as aids in diagnosis. However, the prognosis depends more on the extent of the lesion than on the type of cell, and biopsy gives no notion of the extent. Paradoxically, a normal bronchoscopic examination calls for exploratory thoracotomy, especially in the presence of suspicious roentgenographic shadows.

VOORHEES, N. Y.

THE TREATMENT OF BILATERAL LARYNGEAL PARALYSIS. H. LOEBELL, Pract. oto-rhino-laryng. **11**:232, 1949.

Loebell, in 2 cases of bilateral paralysis of the vocal cords, resected one vocal muscle subcutaneously and followed this procedure by dilation, with either a stuffed rubber finger cot or a Uffenorde rubber tube. In 4 additional cases he supplemented this procedure with a submucous resection of homolateral arytenoid cartilage. Because of the splendid results obtained with this procedure he states the opinion that this is the method of choice.

PERSKY, Philadelphia.

Nose

INTRANASAL APPLICATION OF BUFFERED TRIPELENNAMINE (PYRIBENZAMINE) SOLUTION IN ALLERGIC RHINITIS. J. BREM and J. ZONIS, J. Allergy **20**:70 (Jan.) 1949.

The authors treated 81 patients with allergic rhinitis by the topical application of buffered tripelennamine hydrochloride (pyribenzamine hydrochloride®) solution, and more than 90 per cent of the patients were reported to have obtained satisfactory or excellent results. A 2 per cent solution was first used but was

soon replaced with a 0.5 per cent solution, because the stronger solution caused local discomfort. No serious local or general effects were noted. Topical therapy was effective when antihistaminic drugs orally administered could not be tolerated because of drowsiness and other distressing symptoms.

The local effects were temporary and the usual allergic diagnostic and therapeutic measures were necessary.

FRIEDBERG, Chicago.

CARCINOMA OF THE NASOPHARYNX: REPORT OF 150 CASES. M. W. SIMMONS and I. M. ARIEL, Surg., Gynec. & Obst. **88**:763 (June) 1949.

A series of 150 consecutive, unselected patients with carcinoma of the nasopharynx was reviewed and studied by the authors while in residence at the Hines Veterans Administration Hospital. For each patient the nasopharyngeal biopsy indicated the presence of carcinoma. The commonest site of origin was Rosenmüller's fossa (17.7 per cent). A characteristic of the disease, namely, early cervical metastases, was found in 125 patients (83.3 per cent) on admission. Only in 20 patients (13.3 per cent) did cervical metastases not develop at some time during the course of their illness. Intracranial extension was demonstrated in 9 of the 37 postmortem examinations made, and metastases below the clavicle occurred in 20 instances.

The commonest initial symptom was swelling of the neck, present in 79 patients (52.7 per cent). Pain in the head or face occurred in 25 patients (16.6 per cent), whereas only rarely were the initial symptoms referable to the nasopharynx. Signs referable to the cranial nerves were noted in 44 patients (29.3 per cent) on admission and in 55 patients (36.6 per cent) during the course of their illness.

It was found that patients waited an average of 3.2 months after the symptoms began before consulting a physician. An average of 6.9 months elapsed before the physician made the correct diagnosis. A period of 10.1 months elapsed before adequate therapy was instituted. Among the 128 patients who died, an average time interval of twenty-seven months occurred from the onset of symptoms until death. With adequate treatment, this interval averaged thirty-one months from the onset of symptoms until death. Without treatment, the average life expectancy was seventeen months.

Of the 78 patients with the determinate cases, only 7 remained free from the disease for five years or more, this proportion producing a net five year survival rate of 9.8 per cent. Those patients who were cured were the ones in whom cervical metastases had developed. No patient with intracranial extension survived the five year period. Treatment of carcinoma of the nasopharynx is exclusively roentgenologic.

FRIEDBERG, Chicago.

Miscellaneous

CONTROL OF AIR-BORNE INFECTIONS IN A NURSERY FOR YOUNG INFANTS. IWAN ROSENSTERN, Am. J. Dis. Child. **75**: 193 (Feb.) 1948.

After an epidemic of enteritis in "The Cradle," Dr. Gladys Dick set up an individual aseptic nursery technic in 1929. From its introduction to April 1, 1946 there was no death due to enteritis contracted after admission. Furthermore, 3,780 infants were admitted, among whom were 52 with enteritis, 64 with impetigo and 10 with gonorrheal vaginitis. Previously, elimination of contact, direct or indirect, had had no effect on air-borne infections; intramural air is polluted by sneezing, coughing and talking. Air conditioning, germicidal lights, mechanical barriers and face masks were experimented with (data on the effective-

ness of the first two measures were published previously). Clinical results obtained over a four year period with air conditioning and irradiation of the "upper air" showed an appreciable reduction in cross infections; air conditioning alone did not prevent their spread. Face masks were worn by all attendants. A table shows the results obtained from using two layer gauze masks and those made from vegetable fiber, pressed paper, cellophane and gauze, and gauze-covered flannel filters. The last type was the most effective. A control study, without masks, was also set up. While bacteriologic evidence was not conclusive, it was corroborative and helpful.

A nurse with a cold was sent off duty when possible. However, the infection was not always reported, and it was impossible to keep many nurses off duty at one time because of a shortage. It is striking that the transmission of infections of the respiratory tract from nurse to infant was so infrequent. Germicidal light barriers and mechanical barriers were both useful. Experimental studies on face masks of varying kinds were still being carried on at the time of the report.

VOORHEES, New York.

IMPORTANCE OF ALLERGY IN EAR, NOSE AND THROAT CONDITIONS. W. C. SPAIN, Laryngoscope **57**:1299 (Dec.) 1948.

Only 10 per cent of allergic conditions are of interest to the otolaryngologist. Included are hay fever, perennial coryza, bronchial asthma and cutaneous manifestations. Drug sensitization is important.

Diagnosis must include a thorough history and physical examination. Skin and laboratory tests are likewise important. Although specific immunization is often necessary for cure of the condition, in many instances mere removal of the offending allergen suffices.

HITSCHLER, Philadelphia.

ARTICULATION TESTING METHODS. JAMES P. EGAN, Laryngoscope **58**:955 (Sept.) 1948.

Egan discusses the procedures found particularly useful in the measurements of the intelligibility of speech used at the Psycho-Acoustic Laboratory for the study of oral communication during the war years. Mentioned in the article are the various kinds of speech units used, their relative value, the available lists of syllables and words, etc., and the methods of conducting articulation tests.

HITSCHLER, Philadelphia.

CLINICAL EVALUATION OF A NEW ANTIHISTAMINIC COMPOUND. ANDREW B. PAUL, ANDREW A. EGGSTON, CHARLES J. GAROFALO and RICHARD J. BELLUCCI, Laryngoscope **58**:1044 (Sept.) 1948.

The new antihistaminic drug thephorin® (NU-1504; 2-methyl-9-phenyl-1, 3, 4, 9-tetrahydro-1-pyridindene hydrogen tartrate) is discussed in detail. In general, very good results were obtained with the use of this drug in the author's series of 301 patients. Approximately 75 per cent of patients with hay fever and allergic rhinitis (nonseasonal) and 50 per cent of patients with asthma were helped. Few side effects were noted, the chief one being stimulation. As with other antihistaminic drugs, there is no curative effect but merely a temporary relief of symptoms.

HITSCHLER, Philadelphia.

ANESTHESIA IN ENDOSCOPY. JOSEPH W. KING and DAVID H. JONES, Laryngoscope **58**:1195 (Nov.) 1948.

Patients are interviewed to determine their degree of apprehension prior to anesthesia. Their previous anesthetic records are determined. The type of preoperative medication and of anesthesia can then be decided on. Sometimes general anesthesia, obtained preferably with ether, is necessary.

Proper preoperative sedation is important. It allays apprehension and allows the use of less anesthetic. King and Jones report the results of using a combination of cocaine and tetracaine hydrochloride (pontocaine hydrochloride®) and Forrestier's solution. With both there were some toxic reactions. The final procedure adopted was that of instilling slowly 3 to 4 cc. of a 4 per cent cocaine solution, 1 cc. at a time at five minute intervals. The intervals allowed time to see any toxic reaction, if one should appear. Relaxation by this method was good.

HITSCHLER, Philadelphia.

PENICILLIN IN THE TREATMENT OF DISEASES OF THE EAR, NOSE AND THROAT. VIRGIL L. PAYNE, Laryngoscope **58**:1216 (Nov.) 1948.

There is a review of the properties and pharmacology of penicillin. The modes of administration and the results to be expected from each are discussed. Reactions are mentioned, and several cases are reported. The best results were obtained by the use of penicillin in beeswax and oil, given intramuscularly. Other preparations of penicillin which may be even better are now available.

HITSCHLER, Philadelphia.

UNDERDEVELOPMENT OF THE MANDIBLE AS A SIDE EFFECT OF IRRADIATION THERAPY. P. KISFALUDY, Paediat. danub. **2**:114 (Aug.-Sept.) 1947.

In a 10 year old patient, radium applied in the oral cavity and high voltage roentgen irradiation in the course of which 2,500 r had been given in fractions to both submandibular regions resulted in an isolated symmetric underdevelopment of the mandible which first could be observed at 13 years of age.

The developmental disturbance is to be ascribed to the deep roentgen irradiation. This assumption is favored by the even alteration of both sides of the bone. The radium treatment, acting on a small area only and manifesting itself at the base of the tongue, would have given rise to a unilateral developmental disturbance.

The liability of the permanent teeth to undergo caries is a striking symptom.

FROM THE AUTHOR'S SUMMARY [Am. J. Dis. Child.].

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

William A. Smiley, M.D., President

Lawrence J. Lawson, M.D., Secretary

Monthly Meeting, Dec. 6, 1948

Headache and Neuralgia of Nasal Origin. Dr. Lawrence R. Boies, Minneapolis.

The nasal space and, in particular, the accessory sinuses are often blamed for chronic head pain when the cause cannot otherwise be easily explained. This fact apparently accounts for the frequency with which various surgical procedures have been performed within the nose and sinuses to relieve victims of chronic head pain. It is important, therefore, for any physician to know something about the mechanism giving rise to this disorder and its many causes.

Headache associated with either decreased or increased intracranial pressure results from traction on or displacement of pain-sensitive intracranial structures and is independent of generalized intracranial pressure changes per se. Brain tumor produces traction chiefly on the large arteries, veins and venous sinuses and on certain cranial nerves. The headache of migraine seems to be due to dilatation and distention of the extracranial and, possibly, the dural branches of the external carotid artery. This is also true of headache associated with hypertension; there is no direct relation to the level of blood or pulse pressure.

Following is a classification of causes and forms of headache in four main groups:

SYSTEMIC DISORDERS OR GENERAL DISEASE CAUSING HEADACHE

- Infectious diseases
- Circulatory disturbances: hypertension, hypotension
- Abnormal states of the blood: anemia, alkalosis, uremia, hypoglycemia, polycythemia
- Gastrointestinal disorders

DISORDERS LOCALIZED WITHIN THE HEAD CAUSING HEADACHE

- Intracranial disease
- Ocular disorders
- Disease of the nasal space

MISCELLANEOUS CAUSES

- Relaxation states
- Caffeine withdrawal
- Cerebrovascular syphilis

INDEPENDENT FORMS

- Migraine
- Histamine headache
- Headache of emotional origin
- Headache from disorders in the neck

The independent forms of headache are often encountered in clinical practice. Some of the numerous reports on the use of histamine in treatment of headache lack convincing proof that the diagnosis of the cause is correct or that the therapy could be unquestioned as the source of relief. Williams' observations on physical allergy as a contributing factor are interesting. The neuralgias must also be included; however, physicians should distinguish between headache and neuralgia. Many classifications of facial neuralgia, for instance, are complicated. It has been found useful to list the neuralgias in six groups:

Trigeminal neuralgia, primary and secondary
Glossopharyngeal neuralgia, primary and secondary
Vidian and sphenopalatine neuralgia
Neuralgia from disturbances of the temporomandibular joint
Atypical facial neuralgia
Rare forms:

(*a*) Geniculate ganglion neuralgia

(*b*) Tympanic plexus neuralgia

(*c*) Great superficial petrosal neuralgia

The pain of vidian and sphenopalatine ganglion neuralgia may have a sudden onset, is severe, localized around or back of the eyeball, radiates to the temple, back of the ear to the occiput and into the neck or shoulder. Vail stated that it represented an irritation of the vidian nerve in its course below the floor of the sphenoid sinus. Sluder described two syndromes from involvement of the sphenopalatine ganglion, one neuralgic and one "sympathetic," in which there are also vasomotor and secretory phenomena. Many neurologists seem unwilling to accept Sluder's explanation, but with newer knowledge of vasomotor disturbances, it is likely that this, rather than sinal inflammatory change, may account for some of the painful and probably all of the vasomotor phenomena. Williams explained sphenopalatine neuralgia, particularly the sympathetic syndrome, on the basis of physical allergy. It seems probable that vidian or sphenopalatine neuralgia can be produced from inflammatory changes within a sinus but that much of the picture would fit into Williams' theory of physical allergy.

In the atypical facial neuralgias, distribution of pain is within a circular area in the vascular supply of the head. Sympathetic phenomena, such as lacrimation, edema, corneal injection, unequal pupils, blurred vision and photophobia, are present in about half the cases. The diagnosis apparently is made when the disorder does not fit into one of the well defined neuralgias. In many cases the condition could be a vasomotor disturbance on the basis of physical allergy.

Laymen, and many physicians, are prone to attribute symptoms of headache or neuralgia to the sinuses. It is probable, however, that mistakes in evaluating the role of the nasal space in the symptom of head pain, particularly a chronic pain, are due to an inadequate history and to lack of information on the problem of headache and neuralgia in general. Every clinician encounters frequent cases of pronounced irregularity of the nasal septum, hypertrophied turbinate bodies and roentgen ray shadows indicating extensive changes within the sinus linings in which complaints do not include headache as a symptom. In contrast, instances of contact or congestive headache are frequently encountered in which the nasal interior shows only moderate abnormality, but in which prompt relief is obtained by simple shrinkage with ephedrine or by cocainization of the area of contact.

Therapeutic tests on which otolaryngologists can rely in cases of acute rhinitis and sinusitis with suppuration are (*a*) simple shrinkage or cocainization in cases of congestive or contact headaches and (*b*) drainage and ventilation in cases of sup-

puration within any of the several sinuses. In cases of suspected vidian neuralgia, instillation of 4 per cent cocaine into the sphenoid sinus will relieve pain; cocainization of the area over the sphenopalatine ganglion will relieve neuralgia of that nerve structure. It has been my experience that when these tests are used in cases of headache or neuralgia of chronic type they are reliable in establishing whether or not the nasal space is an etiologic factor in the pain.

DISCUSSION

DR. JOHN R. LINDSAY: I have enjoyed listening to Dr. Boies' presentation of this problem. In his usual logical and clearcut way he has given a very good résumé of the whole situation. I have only one or two additional comments to make.

We physicians know that because the nasal sinuses are sometimes the origin of severe headaches most patients with headaches of obscure origin eventually are sent to an otolaryngologist. The state of affairs now differs from that of ten or fifteen years ago when the cause of these severe headaches was usually considered to be in the nose. One of the faults in former days was the failure to take an adequate history. The more recent recognition of the part played by emotional states and vasomotor disturbances has done much to change the tendency toward unnecessary operation on the nose. We are all familiar with patients who, having pain in the head or face, have had an ethmoidectomy, fenestration of an antrum, removal of the turbinate bones and the teeth, and who still have pain. A careful history and evaluation should avoid such unnecessary surgical procedures.

In many cases the response to intranasal treatment is not reliable. In some cases almost any intranasal treatment seems to afford temoprary relief from headache. Sometimes it appears that one is dealing with a reflex action in response to pain, irritation or vasoconstriction in the nose.

I was interested to note that Dr. Boies finds that the classic histamine headache occurs rarely. Sphenopalatine neuralgia is also a rare occurrence. In some cases we still make injections with alcohol, usually because the neurosurgeon wishes us to do so, but such cases are rare.

Treatment and diagnosis of headaches in general have improved in recent years so far as the rhinologist is concerned, but it must be admitted that there is still a great deal to be done. While we recognize that headaches in some cases are vascular in type, we do not always have an effective treatment to offer.

DR. ROBERT LEWY: About three years ago the Academy had a panel discussion on headache. One cause discussed at that time was sensitivity to light, associated with vitamin A deficiency. I have had occasion to make that diagnosis in several cases, in each of which the headaches ceased on daily administration of large doses of vitamin A. I should like to have Dr. Boies' opinion on the role played by vitamin A deficiency.

DR. WILLIAM A. SMILEY: I should like to ask about vasomotor disturbance in migraine. Is it vasoconstriction or vasodilatation? The ophthalmologists have stated that when migraine is present the arteries of the eye are like a cord. If the vasomotor disturbance is one of vasoconstriction, how can we physicians reconcile our use of ergotamine tartrate U. S. P.?

DR. LAWRENCE R. BOIES, Minneapolis: In answer to Dr. Lewy, I know nothing about light sensitivity as a cause of headache. I have heard ophthalmologists mention light sensitivity associated with vitamin A deficiency and can believe that this sensitivity could well be a cause of a certain amount of headache.

Dr. Smiley asked about vasomotor disturbance in migraine; I mentioned the work of Wolff and his co-workers, who have contributed experimental evidence

to show that the headache of migraine arises chiefly from dilatation and distention of certain branches of the external carotid artery. It is increased amplitude of pulsation in a terminal branch, such as the temporal artery, which causes the pain. The effectiveness of such preparations as ergotamine tartrate in relieving the headache lies in their action in reducing the amplitude of the pulsations of the cranial vessels, which are abnormally active. I believe that constriction of retinal vessels is not an important diagnostic sign in migraine, although I know it has been observed in some cases. The retinal arteries are branches of the internal carotid artery, and I presume vasoconstriction could occur in these vessels along with vasodilatation in certain terminal branches of the external carotid artery.

Dr. Shapiro asked about the significance of vasomotor changes in the nose. I know from my experience that many factors may play a role in vasomotor changes associated with nasal congestion. Nasal congestion is, of course, extremely common and might, in occasional cases, be associated with cerebral congestion; vasomotor changes in the nose might occur in certain circumstances, such as histaminic cephalalgia, but oftener it is a more localized phenomenon in the nose. Headaches resulting from congestion localized in the nose are relatively mild. The therapeutic tests of simple shrinkage or cocainization as a means of relieving the headache are in my experience quite reliable in determining whether or not the source of the headache actually originates in this congestive change. I am not averse to prescribing vasoconstrictors on a temporary basis for relief.

Congestive changes in the nose are common in systemic aspects of endocrinal activity, although there is nothing about these congestive changes which are characteristic. The commoner occurrences are seen both in hyperthyroidism and hypothyroidism, at the menses in some women and at adolescence.

Function of the Ear in Health and Disease: Sound Motion Picture. Dr. Heinrich G. Kobrak.

Otologists celebrate this year the one hundredth anniversary of Helmholtz' work. Since that time, all work on the function of the ear has been based on, or at least influenced by, the fundamental work of Helmholtz. Notwithstanding its many contradictions, and in spite of its numerous unexplainable findings, the Helmholtz theory of hearing is still the leading one on the function of the ear. Present day teaching of the hearing mechanism is based not on direct observation but on the interpretation of anatomic studies and psychologic findings.

Discussion of the merits or faults of the Helmholtz theory is not the purpose of this presentation. The history of physiologic research has shown again and again that theories become unnecessary if and when an ample amount of experimental evidence becomes available. One hundred years after Helmholtz, it appears apropos to review the available experimental methods.

Based on these conceptions, the motion picture camera was chosen for the recording of acoustic vibrations within the ear. The film records with a minimum of interference from the delicate tissues; no direct contact is made with the structures. Once the phenomena are recorded they can be projected on the screen, and thus new details, which may have been overlooked in the initial observation, may be discovered.

Slides were shown demonstrating that the knowledge of sound conduction in the human ear is limited. The nature of the forces which cause vibrations in the ear were so unintelligible to the artist that in the past he has been led by his imagination to create new and strange forces in the ear.

William A. Smiley, M.D., *President*

Lawrence J. Lawson, M.D., *Secretary*

Monthly Meeting, Feb. 7, 1949

DR. FRANCIS LEDERER: The work to be presented tonight by the group from the Veterans Administration Hospital, at Hines, Ill., covers 1,325 operative procedures performed by members of the staff and represents many interesting phases of otolaryngology. Otolaryngology is being recognized as a head and neck specialty and is well supported at the Veterans Administration Hospital. This report will be interesting to the members of the society, as the group is really part of the society, and since there are many veterans here and many members of the Deans Committee at Hines.

Rehabilitation Surgery in Gunshot Wound of the Neck. DR. STANTON A. FRIEDBERG.

This report deals with the rehabilitation of a wounded soldier. Although the numbers of men with service-connected surgical disabilities still under treatment at Veterans Administration Hospital are decreasing, this particular case history doubtless has its counterpart in many other hospitals throughout the country. It is presented in order to illustrate one aspect of veterans' care which inevitably receives less notice with the passage of years following a war.

The patient, a 37 year old infantryman, was wounded during the fighting in the Siegfried Line on Feb. 3, 1945, receiving injuries in the neck and larynx from a shrapnel fragment. A tracheotomy was performed immediately at the battalion aid station and he was evacuated through various channels until he reached the Veterans Administration Hospital, on May 15, 1945.

At his first admission to the hospital, the portal of entry was indicated to have been the left hyoid region; the point of exit of the fragment was in a comparable location on the opposite side. Both sites were healed and the tracheotomy opening was functioning well. There was paralysis of the left hypoglossal nerve and pronounced scarring of the posterior pharyngeal wall. Distortion of the larynx obscured detailed examination so that the vocal cords were not seen. Roentgenograms of the neck showed some fragmentation of the hyoid bone and of the thyroid cartilage. (Slide) There were no foreign particles. The patient was discharged on June 3, 1945, without further treatment.

He was readmitted on Feb. 4, 1946, at his own request, for removal of the tracheotomy tube. A report of a direct laryngoscopic examination at this time mentioned strictures of the aryepiglottic folds and piriform sinuses, with fixation of the right vocal cord. The tracheotomy tube was removed but had to be replaced after several days. Closure of the tracheal stoma was thus deemed inadvisable, and the patient was again released, this time with recommendation for follow-up examination in six months.

He entered the hospital again in February 1947, just two years after the original injury. Two months previous to this admission the wound in the left side of the neck had begun spontaneously to discharge clear fluid. Shortly thereafter ingested liquids escaped through the opening. (Slide) Difficulty in swallowing any sizable bolus was mentioned. The voice was rather rough. Mirror laryngoscopy disclosed severe distortion of the pharynx, extending inferiorly from the base of the tongue. Extensive irregular scar tissue in the pharynx in a series of steplike webs produced a funnel-shaped passage, which narrowed down to a diameter of approximately 1.5 cm. in the hypopharynx. Because of this narrowness no satisfactory evaluation of the larynx could be made with the mirror. The

strictured pharyngeal passage was dilated with a small anterior commissure laryngoscope until the larynx came into view. It then became evident that a web of scar tissue occupied the greater part of the glottic space, with only a small horizontal aperture remaining posteriorly. There was complete fixation of the left vocal cord and limited motion of the right. As the laryngoscope was removed, a probe introduced into the cervical fistula could be seen to emerge in the central portion of the scarred hypopharynx. Roentgenograms of the chest, serologic tests and routine laboratory studies revealed no abnormalities.

It was felt that an attempt should be made to repair the pharyngeal fistula before undertaking restoration of laryngeal function. The extensive mucosal scarring in the pharynx was believed to present an obstacle for satisfactory closure, and consultation for plastic surgery was requested.

The operation on the pharyngeal fistula was performed on May 14, 1947, by Dr. C. W. Monroe, I assisting. General anesthesia was used. A small probe was sutured into the fistulous tract and the tract was dissected out, along with considerable surrounding scar tissue. A portion of the submaxillary gland was involved in the tract and was removed during the dissection. The pharyngeal defect was closed with interrupted dermalon® sutures and reenforced with 00000 chromic surgical gut. A feeding tube was introduced, the remainder of the submaxillary gland and surrounding scar tissue were removed and small rubber drainage tubes were inserted during closure. Because of extensive fibrosis this procedure entailed five hours of painstaking dissection. Primary healing resulted.

Eight weeks later the laryngeal web was excised and laryngeal dilators through size 24 were easily passed. Dilation was carried out at weekly intervals thereafter. This procedure had an added beneficial effect in that the repeated dilations served to increase the diameter of the narrowed hypopharynx. Six weeks after this treatment there was free mobility of the right vocal cord, some limitation of movement in the left cord and a sizable glottic aperture with epithelized margins of the cord. The tracheotomy tube was completely corked for two weeks.

On Aug. 22, 1947, with local anesthesia, closure of the tracheostomy was undertaken. A quadrangular series of connecting incisions were made 4 to 5 mm. above and below the stoma. After the skin was undermined almost to the edges of the fistula, the lateral margins of the prepared flaps were inverted and sutured in the midline with fine surgical gut. This procedure resulted in an epidermal lining for the tracheal lumen and a subcutaneous layer of tissue externally. Two sliding skin flaps of unequal length were developed and brought together so that the line of closure would not fall directly over that of the fistula. (Slide) Care was taken to avoid tension in the wound closure, and several lengths of silkworm gut suture were laid horizontally along the entire length of the field for drainage. The wound healed primarily. There was no evidence of a narrowed airway. The voice was rough but of adequate intensity. The patient left the hospital two weeks after the operation, or almost five months after admission.

He has returned to his prewar job in a paint shop and, despite rather strenuous work, has had no difficulty in performing his duties. His only complaint is inability to raise his voice above the din of particularly noisy surroundings.

The breakdown of pharyngeal scar tissue with resulting fistula two years after the original injury merits comment. The increased propulsive efforts required for swallowing in a fibrotic narrowed hypopharynx may have been responsible for this breakdown. Partial removal of scar tissue and dilation augmented the caliber of the pharyngeal lumen. In all probability, the glottic area was not involved in the penetrating wound, the greater portion of the thyroid cartilage having remained intact. More extensive surgical procedure for establishment of an airway would

have been necessary had there been greater damage to the body of the thyroid cartilage. Release of intracordal scar tissue resulted in free mobility of one cord with partial motion of the other, and dilation was effective in maintaining patency until healing occurred.

The method employed for closure of the tracheostomy is applicable only to relatively small tracheal defects. Larger openings may require the use of skin flaps in which have been incorporated bone or cartilage implants for maintenance of the tracheal lumen.

DISCUSSION

DR. JOHN J. BALLENGER: When I was overseas I saw a case somewhat similar to this one. Of course cases could not be kept track of at that time. My colleagues and I thought we could prevent the cicatricial contraction of the larynx with an acrylic implant, and we inserted one and sent the patient on his way about three weeks later.

DR. FRANCIS LEDERER: Performing a tracheotomy was always a difficulty in war casualties. The placement of a tracheotomy was never uniform. One always feels that hindsight is better than foresight, and when my associates and I finally got such patients after they had gone through various centers, we could not be too critical. However, it did seem that better tracheotomies should be taught to the medical officers, as well as to those physicians in civilian practice.

DR. STANTON A. FRIEDBERG: I should like to thank Dr. Ballenger and Dr. Lederer for their discussions. As a rule, the acrylic implants mentioned by Dr. Ballenger are used in the later stages of reconstructive surgery of the larynx. It would be interesting to know how these implants affect the progress of events when used immediately in laryngeal trauma. It is entirely possible that they might provide a lumen which would heal without the formation of scar tissue contractures.

Management of Advanced Cancer of the Larynx. DR. MAURICE F. SNITMAN.

Malignant disease of the larynx can be divided into two groups: A, the generally curable, and B, the generally incurable. Group A can be divided into two subgroups, which I call A1 and A2. In group A1 are the early cordal lesions amenable to laryngofissure and radiation therapy and not infrequently eradicated by excision biopsy. Pathologists have expressed the opinion that simple stripping of the mucosa would result in cure of such lesions. The response of lesions in this subgroup to almost any type of therapy is so good that it merits no further consideration. Group A2 includes the more extensive endolaryngeal lesions that can usually be eliminated by either laryngofissure or laryngectomy and, in some cases, by irradiation.

Group B includes the so-called advanced and inoperable carcinomas of the larynx. This group may be divided into three subgroups: B_1, that of carcinomas with no cervical metastases but with the primary lesion spread to the pharynx; B_2, that of carcinomas with operable cervical metastases, and B_3, that of carcinomas residual after a complete course of irradiation.

Statistic conscious therapeutists vie for cases in group A_1 and for the more select cases in group A_2. Concentration on this group does little more than inflate the ego of the therapeutist. The problem to be faced is that of group B. This group includes at least 75 per cent of carcinomas of the larynx. The prolongation of life and alleviation of distressing symptoms in cases in this group is the responsibility of the cancer therapist.

In assessing the value of treatment in such cases it is essential to distinguish between immediate and ultimate results. Ultimate results, as reported, have been

universally poor (5 to 10 per cent rate of cure) whether treatment was by irradiation or operation. Reports of the combination of both modalities are very scant and do not supply the clearcut description of the technic and the factors of such therapy, to say nothing of their results. With reference to results in terms of a five year cure rate, my associates and I are not as yet in a position to offer such statistics. We are more concerned with immediate effects and the hope of increased salvage in such cases so as to increase the ultimate results. During the past eighteen months we have modified our standard of operability.

In group B_1 are those carcinomas of which there is no clinical evidence of metastases to the lymph nodes, but of which the primary lesion of the larynx has spread to the pharynx or broken through the laryngeal cage to involve the extralaryngeal structures. If this has occurred grossly, the lesion can be circumscribed by total laryngectomy or laryngectomy combined, if necessary, with pharyngectomy; such a lesion is considered operable. After operation a cancerocidal dose of external radiation is applied.

Carcinomas in group B_2 are the same as those of group B_1 but with operable cervical nodes. The removal of the larynx, with or without a pharyngectomy, is dictated by the extent of the lesion and is followed in ten days to two weeks by radical dissection in the neck, this dissection to be followed by a full course of roentgen irradiation several weeks later.

In group B_3 are the local carcinomas residual months or years after a complete course of irradiation. It has been our experience on repeated occasions to observe such carcinomas. Total laryngectomy performed in such cases is attended by no appreciable alteration of the surgical procedure or of the eventual postoperative progress.

The broadening of the field for surgical intervention and a closer cooperative effort with the radiotherapist has several advantages. The salvaging of some cases, if even a few, more than justifies the effort. Prolongation of life is difficult to estimate and impossible to prove; nonetheless, we are of the opinion that we have prolonged some lives. The alleviation of distressing symptoms has contributed to the comfort of those who could not be cured.

In conclusion, we recommend to the laryngologist a greater concentration and effort in the less hopeful cases of carcinoma of the larynx, to the end that we physicians may achieve more years and more comfort for our patients.

DISCUSSION

DR. JOSEPH G. SCHOOLMAN: I should like to compliment Dr. Snitman and the group at Hines for their work in this field. Those of us who follow patients with cancer—and we tell them we shall follow them the rest of their lives—realize that so many of them may go through a disheartening period. Procedures of the kind described, which can show results in only a relatively small group of cases, do not mean a great deal from the statistical point of view, but from the point of view of the patient they are most important.

DR. FRANCIS L. LEDERER: The formidable enemy cancer of the larynx requires "all-out" effort against it on the physicians' part. Carcinomas in group A are generally amenable to irradiation or to operation. The importance of treatment of carcinomas in group B is to be emphasized. This group should occupy a good part of our attention. I, for one, am willing to disregard statistics. If I have contributed to the rapid passing of some of my patients in these circumstances, I am perfectly willing to take the responsibility, because I do not think that this problem will be solved merely by admitting that such patients are inoperable.

Dr. Figi, at the last meeting of this society, said one could conclude only that all factors covering surgical and medical treatment must be known to the laryngologist. The radiologist is not particularly enthusiastic about treating the patient deemed inoperable, but surely a combined attack, with both modalities, may be responsible for salvaging a few of these patients and may be well worth while.

Otologic Effects of Streptomycin: Supplementary Report. DR. LINDEN J. WALLNER.

In February 1948, I presented before this society a report of aural examinations of 93 patients who received streptomycin for one hundred and twenty days. (Wallner, L. J.: The Otologic Effects of Streptomycin Therapy, ARCH. OTOLARYNG. **49**:108 [Jan.] 1949.) Of those who received 2 Gm. per twenty-four hours, 47 per cent showed objective evidence of vestibular damage; 32.5 per cent of those who received 1 Gm. per twenty-four hours presented signs of impaired function. At that time it was stated that the dose was being reduced to 0.5 Gm. per day in the hope of reducing the toxic effects.

This report is on 66 patients who have received the 0.5 Gm. dose in treatment of tuberculosis, at the Veterans Administration Hospital at Hines. Thirty-nine patients received the dose of 0.5 Gm. for one hundred and twenty days. Later, the drug was given for forty-two days, and 27 patients received it for this shorter period.

Tests on the ear were carried out in the manner described in the previous report. Hearing was tested on the audiometer before, during and after treatment. Caloric tests by the Kobrak method as modified by McNally were done also before, during and after treatment. Subjective complaints relative to the ear were noted. Some of the patients with vestibular damage discussed in the previous report were still available for further study. Tests were done to determine if there was any late return of function in those who had shown depressed or no caloric responses.

RESULTS

Subjective Complaints.—Only 7 patients admitted on questioning that they had noticed some vertigo while receiving the drug. In only 2 patients was it severe enough to affect walking. This lack of vertigo, tinnitus and blurring of vision was in sharp contrast to the presence of these symptoms in the large number of patients while they received the larger doses.

Hearing.—None of the patients had any loss of hearing, as revealed by audiograms made before and after treatment.

Caloric Tests.—Only 2 of the 66 patients (3 per cent) had significant changes in the caloric responses after treatment. These were the 2 patients who had complained of severe vertigo. Each had a lack of caloric response in one ear, noted one month after the drug was started. This lack of response in one ear has persisted to the present time, though neither patient notices vertigo now or has any trouble in walking.

Follow-Up Observations of Patients Previously Reported On.—No patient with lack of caloric responses revealed a return of function when tested later. With some, two years have elapsed since they received the drug.

COMMENT

While 2 of 66 patients, or 3 per cent, presented objective evidence of damage from the 0.5 Gm. dose, in each, only one ear was involved. These 2 patients do not have difficulty in walking comparable to those patients with bilateral damage.

The 0.5 Gm. dose per day is thus seen to be relatively much less toxic to the ear than the 1 or 2 Gm. dose. The first question, then, would be whether this smaller dose is as effective therapeutically. In observing the effects of the drug in laryngeal tuberculosis, I received the impression that the larger doses acted more dramatically. Levine, Klein and Froman stated that the 0.5 Gm. dose is adequate, yielding as good results in pulmonary tuberculosis as larger doses. This is also the opinion of most of those on the tuberculosis service with whom I have talked. Two or 3 Gm. per twenty-four hours is often given for seven to ten days for tularemia, renal infections and other diseases due to gram-negative organisms. If the safer 0.5 Gm. dose is effective in tuberculosis, it might also be as useful in other infections. The newer drugs, dihydrostreptomycin, aureomycin and chloromycetin, are said to be less neurotoxic and just as effective as streptomycin and may provide the solution to the problem.

It is often said that patients soon recover from or compensate for vestibular damage due to the toxic effects of streptomycin. It is true that most patients do not complain of dizziness one or two years after streptomycin treatment and state that they have less trouble walking. Two patients with no caloric responses whose gait was recorded in moving pictures one year ago were again photographed. They are still seen to walk with the feet wide apart, obviously relying on visual aid. When blindfolded, they are quite as ataxic as they were a year ago.

CONCLUSIONS

The toxic effects of streptomycin on the inner ear are serious. When once present, they are likely to be permanent. The 0.5 Gm. dose seems to be a good compromise; it is relatively nontoxic to the inner ear, yet is effective therapeutically. (Slides)

DISCUSSION

DR. SHERMAN L. SHAPIRO: I do not know whether or not I was one of those who thought last year that the toxic effect would be temporary. I certainly do not think so now. This situation, which we physicians hope will be just a landmark in medicine (as well as hope that the newer drugs will not produce such distressing symptoms), does point to a basic fact in medicine. We all know from observation of persons with tabes in days gone by that at least two of three essential senses, the vestibular visual and tactile, are necessary if station and locomotion are to be effective. Even a fish, if deprived of its labyrinth, will swim upside down until it reaches the side of the tank; whereupon, its tactile sense coming into play, it will turn over and swim upright.

I wonder whether these experiments had been carried out in cases of congenital or acquired deafness. It seems to me that the Illinois State School for the Deaf, at Jacksonville, might be a good place to try these experiments to see whether some of the children react in the same way. I think they would. We are dealing here with the basic fact that a person cannot operate successfully with the tactile sense alone.

DR. ELMER W. HAGENS: Several years ago my associates and I examined children in the school at Jacksonville, as well as children in other state schools for the deaf. As I watched the film, I wondered why we had not noted staggering in the children we examined. We did not blindfold them, but certainly they had no such complaints as these patients who had been given streptomycin. So far as I know, it is believed that the pathologic condition in cases of streptomycin toxicity is involvement of the vestibular nuclei at the base of the brain. In the children in the school at Jacksonville the pathologic condition is peripheral. In patients

with meningitis it is a nerve-labyrinth lesion. It may thus be that the pathologic condition in the cases of streptomycin toxicity is involvement of the basal nuclei, and in the deaf children it is peripheral. It occurs to me that the children in the schools for the deaf might be investigated to see if there is any comparison or any contrast. One would think that if the patients presented in the report had no response to caloric tests they would get over this condition. We have always thought that there was compensation; not that such patients could walk well in the dark, but that they should not stumble as these patients with streptomycin toxicity do.

Dr. Marvin Tamari: This film shows ataxia as seen in cases of cerebellar lesions very well. It is known that after operation on the labyrinth or fenestration patients do not stumble or show ataxia, after such a long period has elapsed. Those patients shown in the film, in my opinion, present evidence of central lesions but not that of peripheral. In the postmeningeal cases at the Jacksonville school this type of ataxia was not seen, although there were some balance difficulties, especially in extending the head backward and in darkness.

Dr. Francis L. Lederer: Some of these patients were influenced by tripelennamine hydrochloride (pyribenzamine hydrochloride®). Walking with the broad base gave evidence that this disturbance was not peripheral, therefore suggesting that it may have been a central disturbance. Postmeningitic patients may have certain difficulties for a time, with later compensation, but the patients with streptomycin toxicity have not recovered their equilibrium.

Dr. Linden Wallner: I cannot answer the question of why deaf children with no caloric responses do not have similar difficulty in walking. Perhaps it is because they lost vestibular function at an earlier age. Also, in cases of deafness the lesion is peripheral, usually, while in cases of streptomycin toxicity it is thought to be central.

Some of the patients with streptomycin toxicity have been seen by neurologists, but I do not have their reports available.

Intravenous Procaine Hydrochloride as an Antitumefacient Agent in Rhinoplastic Surgery. Dr. Robert B. Lewy.

Esophageal Varices. Dr. Harkishen Singh, New York.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Louis H. Clerf, M.D., *Chairman*

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Monthly Meeting, Jan. 19, 1949

Localized Osteitis Fibrosa Cystica of the Frontal Bone: Report of a Case. Dr. Philip A. Marden.

A white woman aged 47, a widow, complained of severe pain of three years duration in the region of the frontal bone. This pain originated between the eyes and radiated over both sides of the frontal region. It was present constantly and interfered with sleep. There was no history of associated disease of the upper respiratory tract or of trauma. A few months after the initial appearance of the

pain in the frontal area, the patient became aware of a small hard swelling at the root of the nose. This swelling slowly increased in size during the next two years. The patient had exhibited a remarkable tendency toward formation of cysts in various parts of the body. Both breasts had been removed for cystic mastitis, and the right ovary had been excised for cystadenoma. Moreover, the left ovary was now definitely cystic.

Examination.—A smooth firm swelling, with a diameter of 1.5 cm., was present in the region of the glabella. Firm pressure over the mass caused pain. Both frontal sinuses were clear to transmitted light. All rhinoscopic and nasopharyngoscopic findings were normal. A tender cystic mass could be palpated in the lower part of the abdomen. No cystic swellings could be found in the long bones. Urinalysis showed a trace of albumin and a few hyaline and granular casts. A complete blood count was normal. Wassermann and Kahn reactions of the blood were negative. The fasting blood sugar was 105 mg. per hundred cubic centimeters. The serum calcium was 9.54 and the serum phosphorus 3.75 mg., per hundred cubic centimeters. Roentgenograms revealed a cavity-like formation at the base of the right frontal sinus.

Operation.—On June 6, 1946 the anterior aspect of the bony swelling was exposed. A series of perforations encircling the tumor were made through the anterior plate of the frontal bone with a small perforating burr. The entire mass with its bony shell was then rocked out with a heavy periosteal elevator. During this maneuver small tears occurred in the mucosa of both frontal sinuses. The interior of each frontal sinus was found to be normal. The postoperative course was uneventful. The wound healed with only a slight depression remaining. Pain in the frontal region disappeared after the operation and has not recurred during the succeeding two and one-half years.

Pathologic Report (Dr. R. C. Horn and Dr. R. B. Eisenberg, Hospital of the University of Pennsylvania).—The specimen was an irregular nodule of bone, with one smooth, slightly convex surface, with over-all dimensions of 1.5 by 1.0 by 1.0 cm. There was an irregular cystic area which filled most of the specimen and contained grayish white soft material. Microscopic: The sections showed a border of dense cortical bone with many slightly irregular trabeculae surrounded by numerous osteoblasts and more than a few osteoclasts. The marrow was fibrous. In the region of the lesion proper was a fibrous stroma composed of vascularized fibrillar to myxomatous fibrous tissue, with here and there a collection of mononuclear inflammatory cells, which tended to be perivascular. The tissue was fairly cellular; the nuclei were small, dense staining and usually oval. An occasional angular form was also seen. The blood vessels were generally lined with one layer of endothelial cells. Within this stroma were small islands of osseous tissue. These islands showed varying degrees of degeneration. Some were lamellated with plump osteocytes; others were amorphous with no lamellas or cells. None were lined with osteoblasts. Foam cells were not seen. The diagnosis was osteitis fibrosa cystica of the frontal bone. Morphologically, this condition might have been either a localized lesion or one focus of a generalized lesion.

DISCUSSION

DR. HARRY SCHENCK: This case report brings to mind that of a Marine who was admitted to the hospital because of headache and frontal swelling. An extensive lesion of both frontal bones was demonstrated by roentgenographic examination. At operation, the anterior aspect of each frontal sinus was found to consist of relatively soft and abnormal bone, resembling osteoma spongiosum.

A disfiguring frontal defect remained, and on return to duty the patient complained that his service helmet caused great discomfort. Unfortunately, the pathologic diagnosis had not been made by the pathologist, and the slides forwarded for further study were lost. The patient was killed in landing operations shortly after his return to duty. This lesion may have been similar to that reported on by Dr. Marden; his case was undoubtedly one of osteitis fibrosa cystica. Spicules of bone were present, and since no mitotic figures were noted, the tissue was not malignant. Occasionally, tumors in the frontal region defy accurate diagnosis, but the one reported on by Dr. Marden can be accurately classified.

DR. AUSTIN T. SMITH: The interesting thing to me about such lesions is that they do not have a distinct identity, either etiologically or pathologically. They are classified largely according to structure, depending on the relative amount of osseous or fibrous tissue in them and whether cystic change has occurred. Exostoses, osteoma, osteofibroma, or osteitis fibrosa, and osteitis fibrosa cystica are often described as separate lesions; but one wonders if they may not all have a common background, with common etiologic factors responsible for their origin. It seems to me that their origin can be explained better on the basis of tissue reaction to trauma or injury than on the basis of a true neoplastic process. The same architecture occurs in generalized osteitis fibrosa cystica, due to tumors of the parathyroid, as was present in the lesions in this case; but here there was no derangement of the calcium or phosphorus metabolism.

Experimental Study of Local Use of Gamma Globulin in Treatment of the Common Cold. DR. WM. J. HITSCHLER, DR. FRANKLIN L. RUTBERG and DR. JOSEPH STOKES JR.

Gamma globulin may have antibodies that act against the common cold. If so, treatment by local instillation might be of value.

At the Pennsylvania School for the Deaf, a carefully controlled study was conducted, with the use of daily local instillations of gamma globulin every two hours, from 8:00 a. m. until 6:00 p. m., in those with a cold. A similar number of controls were included in the study. In order to avoid bias the examiners were kept in ignorance of which children were being treated and which were controls. Examinations were made daily. The character and duration of the colds were noted. In addition, the duration of colds in those who had had their tonsils and adenoids removed and in those who had not was recorded.

The duration of the common cold in the treated and in the control group proved to be almost identical. The duration of the cold in those children whose tonsils and adenoids had been removed was less than in those whose tonsils and adenoids were present. The difference, however, was not of great significance statistically.

DISCUSSION

DR. JOSEPH STOKES JR.: The value of this method of study of infections of the respiratory system in the investigation of this type of problem was emphasized. Only in closed populations can adequate control exist for the satisfactory interpretation of clinical observations.

DR. HARRY SCHENCK: Was the gamma globulin used in this study obtained from pooled plasma collected in the Philadelphia area, or was it prepared from plasma obtained from all parts of the country? It is possible that gamma globulin obtained from pooled plasma collected in the Philadelphia area might possess

immunologic properties either absent or in minimal concentration in other parts of the country. Since the bacterial flora of the upper respiratory tract varies widely in different urban areas, it is likely that the immunologic response may be somewhat different in each area. Plasma from one urban center may produce gamma globulin of greater potency for that center than for a center five hundred or one thousand miles away.

DR. JOSEPH STOKES JR.: The gamma globulin used in this study was a polyvalent globulin and was obtained from various cities, including Philadelphia.

Tuberculosis of the Larynx and Tracheobronchial Tree. DR. JOHN J. O'KEEFE.

In order to establish the rationale of the use of streptomycin in tuberculosis of the larynx and tracheobronchial tree, and to support the results obtained by its use, preliminary dissertation and evaluation are made of the therapeutic agents employed prior to the advent of streptomycin. The study correlates the etiology and relative incidence of tuberculous laryngitis with tuberculous bronchitis, both occurring as direct effects of the dissemination of infected bronchial secretions.

Streptomycin exerts an arresting influence on the local mucosal lesions, as well as an inhibitory action on the primary pulmonary lesion. It is because of this combined activity that streptomycin therapy has resulted in such a high incidence of repair in tuberculous laryngitis and bronchitis.

DISCUSSION

DR. GEORGE LIEBERMAN: In the past two years my colleagues and I have tried streptomycin in treatment of patients with laryngeal tuberculosis. Among 22 patients to whom streptomycin was given, 9 improved, 7 improved slightly, 3 died and 3 remained unimproved. Patients improve best when they are free from dysphagia, so that they are more in condition, mentally as well as physically, to be cured of this disease of the lungs.

DR. WILLIAM A. LELL: Dr. O'Keefe is to be congratulated on the excellent presentation of his paper. There is no question that in the past there has been a definite incidence of laryngeal tuberculosis, which has varied according to different investigators and has ranged between 15 and 22 per cent. This variation could well be attributed to seasonal changes, in which an acute infection of the upper respiratory tract may have produced changes in the larynx simulating laryngeal involvement in a patient with pulmonary tuberculosis. Undeniably, there is a definite relation between the degree of pulmonary involvement and the possible laryngeal complications. Patients with extensive pulmonary lesions are more likely to have extension of the infection to the larynx than those with minimal lesions. Again, there is a definite difference in the nature of the involvement of the larynx. Some patients with laryngeal involvement have an infiltrative type of invasion; others, a granulomatous accumulation. Patients with the latter type seem to respond much better to streptomycin therapy, according to my experience, although all patients with laryngeal involvement by tuberculosis show some definite general evidence of improvement with streptomycin therapy.

In cases of tracheobronchial tuberculosis the results appear entirely different with streptomycin and not constant—in fact, in some cases streptomycin appears to have no effect whatever. In many of these cases a simple topical application of silver nitrate solution (10 to 15 per cent) often produces much better results. The lack of response to streptomycin therapy in some cases of such tracheo-

bronchial lesions may be due to the fact that these changes may be associated with an extensive pathologic process, involving not only the mucous membrane but also the submucosal structures with extensive secondary fibrosis; and thus this often becomes an irreversible process.

DR. LOUIS H. CLERF: My interest in tuberculosis of the larynx and the tracheobronchial tree began long before streptomycin was discovered, much less used medically. Then, diathermy was a common form of treatment and silver nitrate, too, was employed; in fact, silver nitrate appeared to give the best results in tuberculous tracheobronchitis. Local treatment of bronchial lesions rarely effected a cure, and cicatricial stenosis was a frequent occurrence.

I have had an opportunity to see a number of patients treated with streptomycin, and I believe that tracheobronchial lesions now can be controlled promptly. I am convinced that it is opening a new era in the treatment of this condition.

DR. JOHN J. O'KEEFE: With relation to Dr. Lieberman's comment on dysphagia, I did not mean to detract from the symptomatic importance of laryngeal tuberculosis. The point I wanted to bring out was that pathologically the effects of bronchial disease are of more importance than are the same changes in the larynx.

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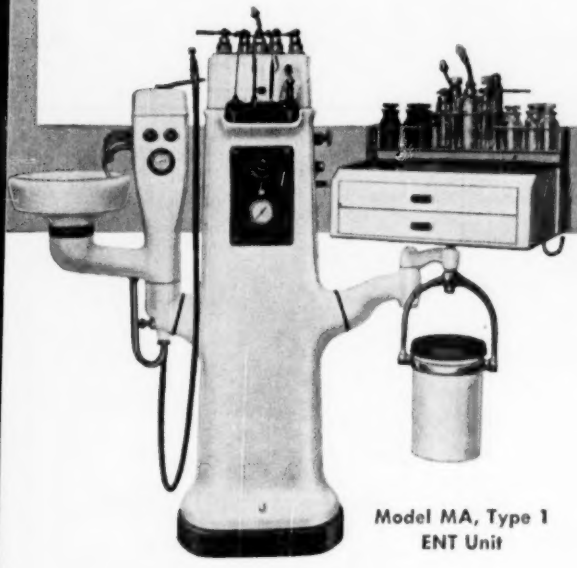

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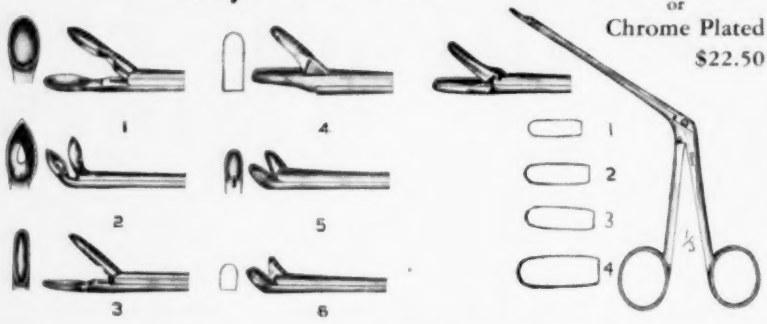

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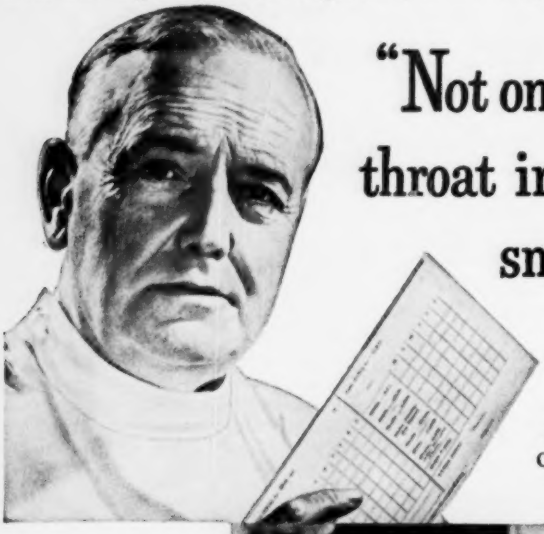

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